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1. Fazekas, J.F., et al.: J.A.M.A. **161**:46 (May 5) 1956. 2. Mitchell, E.H.: J.A.M.A. **161**:44 (May 5) 1956.

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ARIZONA MEDICINE
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Original ARTICLES

AGING LUNGS

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LUNGS BEGIN to age chronologically at birth, and the anatomy and physiology change at an uneven rate from then on.

Actually the potential for change is present before birth. We are not born equal, since the modifications of heredity leave some people less 'equal' than others. They are then prone to the ills and weaknesses of their forebears, or the influence of longevity.

The chance which dictates color of skin, sex, place of birth, and even family type and occupation thus dictates many extrinsic factors which can influence aging of the lungs.

During life we then proceed to mistreat our lungs in a variety of ways – for pleasure, by occupation, or for sport. They have a great resilience and reserve, however, and are able for a long time to fight off most abuse and neglect. . . . Medical science, if given a chance, is able to protect and rejuvenate them somewhat against travail and time, using antibiotics, chemicals, hormones, machines, gases, surgery, etc.

I intend to review some of the things which should be known about the lungs and aging. This is partly for a selfish purpose, so that I may refurbish my own concept of the subject. . . . I do not intend to begin with the infant and work on thru life, but to consider lungs which have had the use of years, and are showing the signs of wear. I will include the premature aging which can occur between 40 and 60 years, and carry on thru the 'struldbrugs' of Gulliver's Travels, with their advanced and once hopeless senility. We might even help the struldbrugs these days.

The changing life expectancy figures, which are so very important in Arizona and Florida and parts of California, should be mentioned. The number of oldsters is increasing. Males are living beyond 65 years, and females beyond 70. . . . There are 12 million people over 65 years of age, which is twice the number of 1930, and 4 times that of 1900. It is 8 per cent of the total population. . . . Disability is 3 times as common between 65 and 75 as at 40 years. Hospital utilization is 4 times as common by retired as by active employees. . . . The life expectancy may rise further, and the population is increasing at the rate of 3 million per year. It is certain that more lungs will grow older, and that more lungs will contain the changes of age and the diseases which occur or accumulate during later life.

The aging of lungs can only be defined by considering changes in anatomy and physiology, due to chronology, infection, occupation, accidents, allergy, and the aging of nerves, circulation, and glands of internal secretion.

Stieglitz says that the **progressive changes of age** include –

1. A gradual tissue dessication.

2. A gradual retardation of cell division, capacity of cell growth, tissue repair, and a reduced antibody formation.

3. A cellular atrophy, degeneration, fatty infiltration, and tissue pigmentation.

Reported to the Arizona Medical Association, Inc., Thurs., May 5, 1955, 4 P.M.

4. A gradual decrease and degeneration of elastic connective tissue.

Examination of the aging lungs must be done in the same way as for any age group, but more emphasis must be placed on objective findings, and the possibilities of the person's age must be used in interpretation. . . . Symptoms and physical signs, both localizing and constitutional, should be obtained. . . . X-rays should always be obtained, remembering that indifference, forgetfulness, emphysema, etc., may minimize complaints and physical findings. It should also be remembered that certain changes take place in the lungs and chest cage from the age of 30 to 70 years. . . . Simple lung function studies may be of value when indicated, and vital capacity, timed vital capacity, and maximum breathing capacity are usually sufficient. Gas exchange studies are in the province of special laboratories. (Dr. Reginald Smart spoke here this winter, and several Arizonans attended a lung funcetion symposium in Los Angeles in February, so I know there is an interest.)

Acute diseases of the lungs include the pneumonias, flareups of chronic infections, vascular accidents, and bronchial lesions. . . . **Pneumonias** have changed in type at all ages in the past ten years. Pneumococcal infections once dominated the incidence compared with other bacteria and viruses; now the situation is reversed. . . . Pneumonia was once called the "friend of the aged" because of its peacefulness. It is quite often bronchial; it may be called atypical, senile, asthenic, hypostatic, etc.; it may mask neoplasms and heart failure; it may accompany arterio-sclerosis, diabetes, bronchitis, and emphysema. Reinmann says that "Enfeeblement and atrophy of tissues is usually accompanied by a decreased ability to react against microbic invaders." . . . The more sluggish reflexes and atrophy of the bronchial elasticity and cilia reduce the barrier to infection and the power to expell secretions. Resistance to certain bacteria decreases with age. . . . The incidence of lobar pneumonia is relatively high in old age, and the mortality is directly proportional to age. Friedlander's, Pfeiffers, diphtheria, coli, and other organisms should be looked for as the cause of unusual pneumonia. . . . The antibiotics have changed the entire picture in the past 10 years. They prevent, abort, or cure the former hopeless lesions, and keep people alive to greater ages. The question of a 'passive' euthanasia must be faced or evaded when the basic condition is hopeless and the treatment drastic.

Acute **embolus and infection** occur more often in lungs of older people. The obese, the cardiovasculars, and the post-ops are susceptible, and 85% occur in patients over 40. More than 85% come from thrombi in the legs. The prevention and management are improved and well-known.

The **chronic lesions** of older lungs include changes which occur as a result of **cardiac and vascular disease; fibrotic lesions, neoplasms, degenerative diseases,** and **chronic infections of the lungs and bronchi.** Each of these categories could require a full-size lecture, so I will scan only the points of recent interest.

The effects of **cardiac disease** are chiefly those of chronic and acute passive congestion. C.P.C. is caused by the late results of rheumatic valvular disease and the increasing defects from arteriosclerosis. They affect respiration by impairing the ventilatory efficiency and the gas exchange, and also produce breathing abnormalities thru the central control mechanism.

Vascular aging in the lungs occurs, but will be discussed with emphysemia. Selective vascular lesions are rare. The stenosis of halophagia and arterio-venous aneurysms are not a special accompaniment of aging. I have recently seen a mysterious fine patchy lesion of one lobe, in a man of 93, rapidly involve both lungs and end fatally.

Fibrotic lesions increase with age. They logically should be due to tissue hypoxia or inflammation. They may be localized or diffuse, and either pleural, peribronchial, interstitial, or alveolar in distribution. It helps to think in these terms, but it is not always possible to make a diagnosis. . . The cases of fibrosis with obvious cause mostly occur with chronic degenerations and infections; the obscure cases fall into a separate group. . . . **Radiation fibrosis** occurs in the age beyond 40 only because the procedure is used for the internal and external neoplasms of that period. Better focusing, plus the rotation method of Dr. Hugh Hare, has reduced its occurrence. . . . The fashionable **chronic 'intestitial' 'cholesterol' pneumonitis** may occur by chance in older lungs and it has no specific cause. Its similarity to cancer and infections may cause uncertainty and excision. . . . The elderly in smoky cities may de-

velop **fibro-anthracosis.** Its scarring is minimal, it does not predispose to other diseases, and it is of no clinical importance. An industrial history is of help in finding or identifying fibrosis from 'dust' diseases, including the inert and mild pneumoconiosis from iron and carbon; the reactive and possibly allergic reaction to sugar cane and cotton; and the lesions from silica, asbestos, talc, diatomaceous earth, and beryllium. . . . The Hamman-Rich syndrome is serious, more progressive, and at present seems to be an entity. . . . Atelectasis should make one suspicious of Ca. rather than simple fibrosis.

Neoplasms of the lung are sometimes correlated with advancing age. The lesions of lymphomata, which are next most common, are not. The occurrence of metastases from other organs depends on the source, with the stomach, prostate, kidney, adrenal, and ovary contributing in that order. . . . Bronchogenic carcinoma is correlated with age, and the incidence is increasing in the older age groups. It is 25 times more common in U. S. and European males than it was 35 years ago, and its greatest frequency (37%) is in the 6th decade, an earlier peak than in the past, and one of the few conditions in which such a rise occurs. . . . Dr. Alton Ochsner, known to many of you because of his visits here, feels that there is an undeniable parallel with excessive smoking. Cigarettes contain a carcinogen. Metaplasia does occur in the bronchi of smokers. Viral pneumonia is not the same as 'atypical,' which can be carcinoma. The chances for a male over 40 to have Ca. is greater than to have viral pneumonia. Eighty per cent of carcinomas can be diagnosed pre-op. Cell studies of bronchial secretions are an added help to other technics. A recent series of 300 cases of pleural effusion in older patients was found to have Ca. in 66%. . . . Operability, resectability, morbidity, mortality, and end-results are nearly the same at 65 to 75 years as at earlier ages, but none of the figures are good. . . . Lobectomy may be the way to reduce operative hazards for isolated, smaller (and thus earlier) lesions in the older patient. . . . The only chance to improve the statistics is early diagnosis. Men who have been heavy smokers for years should have a closely-read x-ray every 6 months. . . . Dr. Ochsner (with a straight face) has noted that cancer takes an increasing number of smokers by 55, tho some are 'saved' by having coronaries (from the same cause) at 45. He suggests that smokers may avoid both by shooting themselves at 40.

The regenerative diseases are the greatest clinical problem, and pathological puzzle. I do not know of anyone who has had the complete insight to catalogue them, and the literature is confusing; there are ten names used for one variety of emphysema. . . . Miller voices the most hopeless feeling when he says that the cause of progressive emphysema is uncertain, all varieties increase with age, and care is difficult. I also am depressed by the notable difficulty in explaining the condition to the patient and family. . . . Emphysema is important to the G.P. because most people over 65 live at home instead of in institutions. Considering the pathogenesis more people are reaching the age of clinical symptoms, will be diagnosed, and will need therapy. There are a dozen major methods of therapy. . . . **Obstructive, hypertrophic emphysema** developes thru the years. Banyai believes that infections cause a degeneration of elastic fibres, a partial check-valve in the bronchioles (often with muscle spasm), and the pressures in the lung are elevated from "a lifetime of coughing." The effects on chest size and mobility, gas transfer, etc., are well known. . . . **Non-obstructive, atrophic, senile emphysema** is generally considered to be a usual process of aging, an involutional defect which increases at 60, is constant at 80. The chest is not large, the alveoli are flabby and collapsable, the air is not trapped, bronchospasm is rare, and there is not much functional impairment. . . . Crenshaw of Oakland has recently stressed his belief that non-obstructive emphysema is a hypertrophic condition, with bulla-formation, and 'cotton-candy' or 'vanishing' areas. This is due to degeneration from an obliteration of the bronchial and pulmonary arterioles. He removes the useless, air-robbing segments. Early diagnosis is helpful. Symptoms, physical exams, PA and lateral x-rays, bronchograms, and observation can usually label the condition.

Pulmonary cysts may be present in aging lungs both coincidentally (if congenital, bronchiectatic, or pneumatoceles), and because of the changes in a complicated emphysema. The latter is the more common. . . . Korol has reported an interesting angle in his 400 cases

of advanced emphysema, – The 40 congenital cystic cases produced 7 cases of bronchogenic Ca. in 10 years, while 60 cases of bullous emphysema had no carcinoma.

Chronic infections of the lungs pile up during the years, and can be bothersome, serious, or fatal. . . . Chronic bronchitis can flare into pneumonia, obstruct into emphysema, and be associated with every lesion of the lungs. The older the patient the more serious its implications. It produces a non-specific restrictive fibrosis as time passes. It is most serious in the debilitated. Prevention of progress is probably easier than eliminating the cause. . . . A recent article on **'Tobacco Bronchitis'** surveyed 4,300 cases. Seven to 19% of non-smoking children and adults had bronchitis; 76 to 80% of smokers had bronchitis. Inhalation of cigarettes was the cause, and it often decreased after the age of 50.

Suppurative pneumonia and abscess are usually the result of aspiration, and may occur in the aged. **Bronchiectasis** probably persists from repeated bronchial infections in childhood, with few symptoms for years, and then recurring infections, ill health, and frequent hemoptyses in later years.

Mycotic (fungus) infections are more common in older lungs, but chiefly as residuals. At that stage the coincident fibrosis and emphysema can be embarrassing. Old lungs may acquire new fungus infections, but they are not as specifically serious from dissemination as was once thought.

Tuberculosis is now predominantly a disease of middle and older age. More cases now exist at those ages. More older cases are being found, with 50% of the new cases in New York being over 45 years of age. The average age of patients admitted to sanatoria (as shown by Paulsen in ARIZONA MEDICINE) is rising; 12% of the patients admitted to Los Angeles' Olive View San. in 1940 were over 40, but last year 55% were beyond that age; and more cases will survive as more are being salvaged by therapy. . . . The median age of death from TB in 1924 was 33 years, and it is now over 50. The highest point of the present mortality rate is in males from 55 to 60 years. . . . Waring, and Smith, believe that the seed-bed of future TB cases is the unrecognized tuberculous male over 40, and woman over 60. . . . They are hard to find on a voluntary basis, tho. Only 16% of people over 65 were x-rayed in a recent Florida survey. They rarely will come to be x-rayed in elderly Pasadena. There should be a bounty paid for x-rays on each person over 65. . . . It is by far the worst disease of old age, since it is not only personally serious, but an infectious hazard. Patients who are infectious should be treated with modified rest, PAS and isoniazid, and, if they continue infectious should be candidates for surgery. 75% of older patients with active TB will be dead in 5 years if untreated. This active therapy is more logical because, as Dr. Howson says, persons over 60 can't be trained in precautions.

The aging lung may sometimes be rejuvenated, or prevented from growing older, whether the change be local or generalized. . . . Resectional surgery may halt various types of destruction, and several reports indicate that operations should be done if the need is great, the condition good, and in spite of the age – actually in spite of coronary disease which does not quite double the usual hazard of 3 to 4 per cent. . . . Intermittent positive pressure breathing, plus bronchodilators and wetting-agents, may help the bronchospasm and infection of chronic emphysema in 80% or more of a series averaging 57 years of age. . . . Breathing exercises may be an adjunct of some value in giving diaphragm control and completing the expiratory phase. . . . Pneumoperitoneum may help some patients with low diaphragms. .. . Drugs which dilate the bronchioles and small arteries may give temporary relief and sometimes control of a chronic situation. . . . Antibacterial drugs can work miracles if well chosen, alternated, and wisely used, but the patient **must** be kept under observation. I say 'antibacterial,' since the sulfa drugs are useful as well as the antibiotics. Sadly, stilbamidine for blastomycosis is the only successful drug for fungus diseases and most virus infections. . . . Diamox is said to be useful for congestive lung diseases, with or without emphysema, but is is toxic. . . . The adrenal corticoid drugs may be wonderful for the bronchospasm of asthma, emphysema, and certain fibrosing lesions. I believe that we must learn to **control** and **continue** steroid usage for best effect. . . . It is possible that the new 1 to 20 estrogen-androgen ratio may help the pulmonary as well as the general

condition of both men and women over 60 years. . . . Climate has a legitimate field in therapy of aging people and aging lungs. They may live more comfortably in a dry subtropical climate, with less hazard of 'strep' and other infections, and with the assurance of good care. I suspect that there are more unpubicized experts in geriatrics (or gerontology) in southern Arizona than in all the specialty journals. . . . I apologize to you, therefor, if I have been redundant in this tour of the subject, but it has been a pleasant trip for me. Thank you.

CEREBRO-VASCULAR DISEASES

Charles A. L. Stephens, Jr., M.D., F.A.C.P.
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CEREBRO-VASCULAR diseases have for generations presented the physician with an almost insoluble therapeutic problem, a mysterious etiology and a nonpredictable course. Unfortunate victims of "strokes" or "apoplexy" have in the past been relegated to the remote corners of the wards or tucked away in nursing homes or institutions to vegetate their few remaining years, removed from their families, in sombre surroundings, alone, helpless, often dumb, and without hope.

This unhappy state of affairs has come as a stepchild of ignorance and of economic necessity. There are few physicians that have not stood by helplessly and watched "poor John" suffer another "stroke" and seen the family savings gradually disappear down the costly drain of domiciliary care.

The real magnitude of the problem of cerebro-vascular diseases has recently been revealed by a survey conducted by the American Heart Association and sponsored by the Lasker Foundation.(1) In 1952, 170,000 persons died of vascular diseases affecting the cerebro-vascular system – three times the number of deaths due to tuberculosis and diabetes combined! Seventy-three per cent of these deaths occurred in persons 65 or older. But looking at it another way: 27% occurred in the highly productive years, under 65, i.e. 44,000 deaths from cerebro-vascular diseases in 1952 were in the working age group of 24 to 65 years. If these had lived even an additional one healthy year they could have earned an average of 151 million dollars and paid the Federal Government 18½ million dollars in taxes! The cost of 60,000 victims of cerebro-vascular diseases in state mental hospitals was, in 1952, 46½ millions; the average length of stay of these patients was four years.

It is estimated that in 1955 there were 1,800,000 victims of a cerebro-vascular disease in the U.S.A.

In St. Mary's Hospital, Tucson, Arizona, in 1955, there were 189 admissions for cerebro-vascular diseases, with an average over-all mortality rate of 50%.

The problem, then, of cerebro-vascular diseases in this country and in this city and to ourselves and the people we care for and assume responsibility for, is a great one – great in numbers and great in cost to us all.

It is the purpose of this paper to define this common problem, to review that which we know and do not know, and to cite some personal experiences in the diagnosis and treatment of cerebro-vascular diseases.

TEMINOLOGY

While it is possible to dismiss the whole matter with the vague and all inclusive off-hand diagosis of "C.V.A.," it is essential that we agree on more specific terms, so that we may find a common basis for communication and the accurate interchange of knowledge.

In a search of the literature (1-20) and in reviewing reports at one of our local hospitals, a variety of terms have appeared. Unfortunately, to all men these words do not have the same meaning. The following is a list of diagnoses used:

stroke
apoplexy
a stroke of apoplexy
shock
ictus
hemiplegia
senile stroke
softening of the brain
subarachnoid hemorrhage
anemic infarct
cerebral arteriosclerosis
cerebral ischemia

*Presented at regular meeting of A.C.P., Tucson Arizona, February 4, 1956.

cerebral thrombosis
cerebral hemorrhage
cerebral embolus
"C.V.A."
focal cerebral infarction
hemorrhagic infarct of the brain
cerebral spasm
cerebral spasm
hypertensive encephalopathy
encephalomalacia

It is obvious that many of these terms are non-specific, redundant and misleading. It would seem appropriate for the Committee on Cerebro-vascular Diseases of the American Heart Association to agree on a definition of terms and recently a subcommittee for this purpose has been created.

PATHOLOGY

The anatomy and physiology of the brain and its blood supply is in many ways unique.(2) The cerebral tissue has an RQ of 1, signifying an exclusive use of glucose for energy. In fact, the brain uses over ten times the amount of glucose per gram than any other tissue in the body. Therefore, when there is circulatory arrest, even briefly, irreversible damage may result.

The brain uses 20% of the total oxygen requirement of the body and respiratory arrest may be as hazardous for the brain as circulatory arrest.

The vascular bed of the brain is rich and multiple but unfortunately effective channels of collateral circulation do not exist* between the various terminal arterial branches supplying the brain. When one appreciates that the brain is highly organized and segregated as to function, it is not difficult to understand how a small but appropriately placed infarct may lead to severe disability or death.

The blood vessels of the brain are subject to the same defects and diseases seen elsewhere in the body, and which may result in infarctions:

ARTERITIS may result from collagen disease, such as lumpus erythematosus disseminatus, periateritis nodosa, polyarteritis; from Buerger's disease; from rheumatic fever; from diabetic or hypertensive arteritis or arteriolitis; syphilis; from mycotic aneurysms, sepsis, trauma, eclampsia.

(*There are collaterals which can take over when occlusion of a vessel is slow,—viz. slow occlusion of a middle cerebral artery may not result in infarct but sudden occlusion does.)

EMBOLI may arise from the heart or from the pulmonary veins. In the heart, rheumatic heart disease with auricular fibrillation or arteriosclerotic heart disease with auricular fibrillation, myocardial infarct with mural thrombi or SBE, may fracture thrombi and lead to embolization and infarction of the brain.

ANEURYSM may be congenital in origin or acquired as a result of arteriosclerosis or hypertension. Their common location at points of 180° angulation implies aneurysmal dilation from a resultant of pressure forces.

ARTERIOSCLEROTIC BRAIN DISEASE may lead to thrombosis in situ in a large vessel with a corresponding large anemic infarct or multiple small vessels may thrombose leaving a pattern of small areas of focal necrosis.(3)

A recent important addition to our understanding or arteriosclerotic brain disease is the concept of **cerebral insufficiency** which corresponds to the older, firmly established coronary insufficiency. There is evidence to support the theory that a patient with cerebral arteriosclerosis may develop in devious ways a cerebral hypotension; this in turn fails to supply sufficient blood to portions of the brain already at a critical level of blood flow and infarct may result.

HYPERTENSIVE ENCEPHALOPATHY with necrotizing arteriolitis and spasm remains something of an enigma. There is still no general agreement concerning the existence of pure cerebral arterial spasm — some maintain that spasm of the cerebral vessels does occur but only around a small thrombosed artery as the initiating insult. Others deny this and feel that malignant(4) hypertension can originate the arterial spasm.

BRAIN HEMORRHAGE at the autopsy table may be seen to arise from arteriosclerosis, malignant hypertension, vascular malformations, or hemorrhagic diatheses (i.e. thrombocytopenia leukemia, etc.) Dr. R. D. Adams of Harvard Medical School (3) states that in his material, "Almost all cases of brain hemorrhage are fatal. We rarely see an old hemorrhage in the brain; whereas, countless old softenings are encountered. Nearly all of the patients were hypertensives and the retinal and brain lesions nearly always parallel each other." Dr. Zimmerman(5) of New Haven Hospital, found 182 patients with cerebral-vascular disease out of 4,240 consecutives; from his examination he agrees that

about 80% of those with brain hemorrhage are hypertensives and it is his belief that the hemorrhage is almost always due to dissecting aneurysm of the fusiform type (Charcot-Bouchard aneurysm). Blood dissects beneath an arteriosclerotic plaque and rupture follows. Dr. Sheinker of Cincinnati General Hospital (6) studied 300 massive cerebral hemorrhages and found 65% in the basal ganglia; 15% in the white matter; 20% in various other areas.

HEMORRHAGIC INFARCT is regarded by most pathologists as a separate entity, the final termination of a series of events. First, there occurs an anemic infarct following arterial occlusion or insufficiency. In sequence the adjacent ischemic tissue becomes necrotic and vessels rupture and bleed.

Disturbing to the pathologist is the disproportion sometimes found between minimal findings at post mortem and the extensive clinical findings described on the patient's record. Unfortunately the pathologist cannot measure and appreciate the critical factors of cerebral blood flow, and other physiological dynamic measurements only present in the living patient.

DIAGNOSIS

HISTORY: It has long been said that "The patient with cerebro-vascular disease usually will not have premonitory symptoms. Rarely will the patient complain of vertigo, weakness, transient weakness of an extremely, ophthalmopelgia, headache, nausea and vomiting. The usual onset is sudden and without warning."(9) As we have become more alert to the subtilities of cerebro-vascular diseases, this no longer applies.

An example is the history of "The Little Strokes" so often and vividly described by Dr. Walter Alvarez.(18) These patients may have brief "black-outs," dizziness or weakness, transient confusion or aphasia, brief episodes of ataxia and gradual personality changes of a permanent nature. Recent studies(15) by Siekert and Millikan of Rochester, Minnesota, lay particular emphasis on the history of recurrent episodes of aphasia, numbness and tingling and weakness of the right arm, due to intermittent insufficiency or thrombosis within the basilar artery system, or intermittent hemiparesis, sensory defects and aphasia due to intermittent insufficiency or thrombosis of the internal carotid artery. C. J. Wood (16) and others (17) have pointed out the episodic history of repeated cerebral emboli from rheumatic heart disease or arteriosclerotic heart disease with auricular fibrillation and intra-auricular thrombi.

General symptoms of cerebro-vascular diseases commonly encountered are headache, nausea and vomiting and coma. Coma is much more common with cerebral hemorrhage.

In Dr. Merritt's series at Columbia University, New York,(7) the following incidence of symptoms were found:

Symptoms	Cereb. Hem.	Cereb. Thr.	Cereb Emb.	Subarach. Hem.
Headache	63%	6%	25%	
Vomiting	51%	6%	23%	50%
Convulsions	15%	7%	9%	15%
Coma	51%	33%	25%	30%

Hemorrhage usually causes a much greater disruption of brain function. Findings such as paralysis of conjugate deviation, bilateral Babinski and a severer degree of coma are more common with hemorrhage than with anemic infarct.

Diganosis is related to age as may be seen in the following table taken from Merritt.(7)

Age	Cereb. Hem.	Cereb. Thr.	Cereb. Emb.	Subarach. Hem.
Under 20	1	0	9	11
20-40	3	1	23	36
40-60	48	31	41	38
60-80	46	61	22	14
Over 80	2	7	5	1

Therefore, if one has a patient whose age is under 40, cerebral embolus or subarachnoid hemorrhage becomes a more likely diagnosis.

PHYSICAL EXAMINATION: Examination of a patient with cerebro-vascular disease should be as complete as circumstances will permit. One should recall that the cerebral circulation may be impaired due to extracerebral factors.(9) The first sign of a myocardinal infarct may be hemiplegia. 4.9% to 29% of patients with coronaries were found to have paralytic strokes by Dozzi.(10) Rogers(11) found in a review of 134 cases of cerebro-vascular disease that these two conditions coexisted in 19 patients (7%). Infarct, cardiac arrhythmias or heart failure, may lead to intracardiac thrombi with cerebral embolization, or to hypotension, and a lowered cerebral pressure with resulting cerebral thrombosis or insufficiency. Therefore, careful examination of the heart, including an **electrocardiogram** should be performed on all victims of cerebro-vascular disease.

The carotid sinus reflex should be tested routinely in these patients. Some will be found to have increased sensitivity because of cerebral arteriosclerosis or because of previous heart damage, and the cardiac arrest and lowered cerebral pressure will cause cerebral infarc-

tion. The same may be said of other hypotensive stimuli, such as shock, ganglionic blocking agents, vasodilators, postural hypotension, anemia. The patient's blood pressure should be taken, when possible, both recumbent and standing. Those with postural hypotension and cerebral arteriosclerosis are prime candidates for cerebral infarcts.

Palpation of the internal carotid artery through pharynx, as described by Dunning,(12) may be of great value in establishing the diagnosis of thrombosis of the internal carotid artery. This procedure assumes greater importance when it is realized that hemiplegia, sensory defects and aphasia, commonly described as due to thrombosis of the lenticulostriate artery in the dominant hemisphere, is due to thrombosis of the internal carotid artery if more than the hemiplegia persists.(9)

Skull films may show a calcific internal carotid artery within the cavernous sinus and angiography may reveal the location and extent of the vascular lesion.

Spinal tap should be performed routinely, but with caution. 85% of patients with cerebral hemorrhage will have blood in the spinal fluid, a rare finding in cerebral thrombosis.(14)

The electroencephalogram may be of aid. Schwab(13) found that 40% of all patients with cerebro-vascular diseases have abnormal electroencephalograms or pneumoencephalograms. Although this is less help than you would get if you tossed a coin, serial encephalograms will often aid in differentiating cerebro-vascular disease from tumor.

The search for other manifestations of thrombo-embolism should be diligently carried out. McDevitt(14) reports 98 patients coming to necropsy with a diagnosis of cerebral hemorrhage, cerebral thrombosis or cerebral embolism. Forty-eight had anemic infarcts of the brain and 23 of these exhibited evidence of 50 major thromboembolic complications occurring other than in the brain. Of 525 autopsies performed routinely by Adams(3) in a neuropsychiatric institution, 4% of all organic brain diseases were associated with recurrent cerebral embolism. Of 6,285 unselected autopsies performed by Garvin(18) 4.2% had mural thrombi in the heart. In 771 autopsies on adults who died of hypertensive heart disease intracardiac thrombi were present in 34.4%. Therefore, many of our patients with cerebro-vascular disease will have evidence of thromboembolism elsewhere. If we search for and find these clues our diagnosis becomes more specific.

The differential diagnostic criteria between the three most common types of cerebro-vascular diseases are listed in the following chart from Wright, McDevitt & Foley:(19)

ONSET	CEREBRAL HEMORRHAGE Severe headache, nausea & vomiting, coma	CEREBRAL THROMBOSIS Weakness of arm/leg, Difficulty speaking Gradual or sudden	CEREBRAL EMBOLISM Very sudden-abrupt development of neurological signs
Convulsions	14%	7%	May occur
Coma	If coma persists more than 24 hrs. hemorrhage more likely	Usually less tahn 24 hrs. Often not at all	Not usual unless embolus is large
Vomiting	51%	6%	25%
Incidence	15%	82%	3% (or more)
Age groups	Elderly	Elderly	Younger
General physical exam.	Evidence of arteriosclerosis Hypertension	Same More likely if B.P. normal	Rheumatic heart disease, auricular fibrillation due to arrhythmia, Myocard. infarct, Evidence of emboli elsewhere
Cheyne-Stokes'	Common	Seldom	Rare
Conjugate deviation of eyes	Frequent	Seldom	Rare
Stiff neck	Frequent	Rare	Rare
Quadriplegia	Rare	RRare except in thrombosis of basilar art.	Rare
Babinski bilat.	Frequent	Rare	Rare
Leucocytosis	More than 50% have over 12,000	Uncommon	Uncommon, unless embolus is infected
Cerebrospinal Fluid			
Color	Bloody or xanthochromic	Clear	Clear, or slightly xanthochromic
Pressure	Up	Normal or under 250mm. H2O	Normal
Cells	R.B.C.—crenated	Slight pleocytosis	Normal to moderate pleocytosis
Pr	Up	Normal to slight	Normal to slight
Mortality	90%	60%	50%

TREATMENT

Brain hemorrhage presents a therapeutic problem that is almost insurmountable. Fisher(20) states, "The treatment of hemorrhage is hopeless. Nine out of ten die." Bucy(21) agrees that hemorrhage is usually too rapidly fatal or too extensive and of those who do survive the first insult 50% will die in the first year. He believes surgical intervention offers the greatest hope for the patient and finds cerebral aneurysm with rupture most amenable to surgery. Evacuation of subdural or epidural hematomata may be life saving, and when hemorrhage is in an inaccessible place ligitation of the common carotid artery may salvage an otherwise hopeless patient.

Cerebral thrombosis or embolus, or arterial insufficiency is more amenable to treatment. A wide variety of cerebral vasodilators are in use today. Kety(22) has evaluated many of these. His findings are based on measurements of cerebral blood flow utilizing nitrous oxide and arterial-venous oxygen differences in normal subjects and in patients with hypertension, arteriosclerosis or Parkinsonism.

He has found **stellate ganglion block** exerts little or no influence on the cerebral circulation. Millikan(24) found no change in the natural course after stellate ganglion block.

Chemical and hormonal influences are of the greatest import on the cerebral circulation, i.e. CO_2.

Hyperventilation will produce a 35% reduction in cerebral blood flow and inhalation of 5 to 7% CO_2 will increase cerebral blood flow by 75%. Fazekas and his associates (23) agree and state, "CO_2 (5%) inhalation seems to be the most efficacious agent available for increasing quantitively the rate of cerebral blood flow by reduction of cerebro-vascular reistance."

Aminophyllin intravenously decreased the cerebral blood from an initial level of 59 to 44 mi/100 gm/min. Therefore, aminophyllin is mildly vasoconstrictive in the brain. Caffeine exerts an almost identical effect.

Noradrenalin raised the blood pressure 29% but decreased cerebral blood flow by 9%.

Epinephirin raised blood pressure 20% and increased cerebral blood flow 20% and there occurred a 22% increase in O_2 consumption.

Nicotinic acid and alcohol did not influence cerebral flow even through flushing of the face occurred.

Papaverine increased cerebral blood flow 20%.

Histamine dilates the cerebral blood vessels but this desired effect is annulled by the associated drop in systemic blood pressure.

Oxygen constricts the cerebral vessels and decreases cerebral blood flow by 13%.

Cerebral stimulants and depressants have almost nothing but harm to offer the victim of cerebral arteriosclerosis. Barbiturates, chloral and most narcotics depress still further the cerebral oxygen consumption, and thereby increase an already critical cerebral anoxia.

Rauvolfia and chlorpromazine may help control agitation, but side effects may limit their usefulness and a Parkinson-like syndrome may result.(23) There is no evidence that metiazol or meratran are helpful as cerebral stimulants(23) in victims of cerebral arteriosclerosis.

Antihypertensive agents are properly in use for hypertensive disease, but in patients with cerebral arteriosclerosis the blood pressure should not be reduced to normotensive levels for cerebral ischemia may result. The ganglionic blocking agents may lead to postural hypertension and a tragic result from the precipitous drop in blood pressure and the accompanying cerebral arterial insufficiency. Dr. David Barr(25) states that he believes that many (even the majority?) of cerebral infarctions occur without organic occlusion of a vessel. Excessive doses of hypotensive agents may tip the scales in a delicately balanced cerebral blood flow.

Anticoagulant therapy is an appreciable advance in the treatment of cerebral vascular diseases. The purposes of anticoagulant therapy in cerebro-thrombosis or embolism have recently been outlined by Wright and his associates:(19)

1. To prevent new emboli.
2. To prevent propagation.
3. To prevent additional thrombi in other vessels.
4. To prevent stasis venous thrombosis from lying in bed.
5. To encourage more rapid disintegration of the thrombus by enzymes which act more freely in the presence of anticoagulants.
6. To promote more rapid recanalization of the thrombus.

The first report in the literature concerning anticoagulant therapy for cerebral vascular dis-

ease appeared in Acta Medica Scandinavica in 1941.(26) Dr. Per Hedevinis reported 26 patients with cerebrovascular disease; of these 18 had cerebral thrombosis and were given heparin with five good results, 13 uncertain affects and three without benefit. Two patients had cerebellar thrombosis and one had a good result and one no effect. Six patients had cerebral embolus and two patients had a good result, two uncertain and two none.

Drs. E. V. Allen and Nels W. Barker of the Mayo Clinic in 1943 were first in the United States to suggest the use of anticoagulants in the treatment of thrombosis of the cerebral arteries.(27)

In the following years numerous anecdotal reports appeared in the literature, (16)(28) both supporting(15) and opposing(29) the use of anticoagulants. The most recent and comprehensive evaluation of prolonged anticoagulant therapy in cerebro-vascular disease is reported by Wright and his associates.(31) A total of 57 patients were observed both on and off of anticoagulant treatment. Thirty-one of these patients had intramural thrombi, either due to rheumatic heart disease with auricular fibrillation, or arteriosclerotic heart disease with myocardial infarction; 19 had arteriosclerosis cerebrae, and seven had hypertension or other cerebral diseases. The results are as follows:

No Rx	57 patients	795 patient months	205 thromboembolic episodes 81 cerebral
Anticoag Rx	57 patients	1162 patient months	23 thromboembolic episodes 6 cerebral

Compared by a ratio on the basis of a thousand patient months the figures would run somewhat as follows: in the treated cases there were 22 total thrombo-embolic in a 1000 patient months. Those same patients when untreated for a 1000 patient months would have had 350 total thrombo-emboli. The treated patients had seven cerebral thrombo-embolic and the untreated patients would have at the same rate approximately 150.

In the author's experience extending over a 9 year period with a total experience in excess of 100 patients on ambulatory anticoagulant therapy(25) 16 patients had cerebral-vascular disease the results are similar.

Millikan and Seibert(16b) have reported favorable experiences in 10 patients and they expressed as late as October 1955(15a) their indications for the use of anticoagulant drugs in cerebrovascular diseases:

1. Intermittent insufficiency or thrombosis within the field of the basilar artery system.
2. Intermittent insufficiency or occlusions of the internal carotid artery.
3. Multiple cerebral infarctions.
4. Prevention of thrombo-embolic phenomena in patients with rheumatic heart disease and mitral stenosis or patients with coronary thrombosis and myocardial infarction.

In occluded arteries in the brain, hemorrhage commonly occurs around the edges (i.e. "hemorrhagic infarct"). This is not a contraindication to anticoagulants.(19)(31) The same phenomenon occurs in infarcts of the heart, lung, kidney. Blumgart studied this problem in animals and found bleeding in all infarcted areas. However, the animals on anticoagulants did not have an increase in blood around the infarctions compared to the controls; neither was healing delayed.

REHABILITATION: Not the final but a concurrent phase of treatment is rehabilitation.(32)

At the earliest possible time, sandbags and splints are applied to keep the exrtemities in the optimum positions. The extremities are put through the full ranges of motion ten to fifteen times, three or four times per day, beginning on the second day. Early ambulation is desirable unless contraindicated and exercises with the use of pulleys are encouraged. For some patients a short leg brace with a 90° stop is a great aid in walking. Constant psychological help is an essential to get the patient to try to help himself and to regain some measure of hope and confidence. Of 1000 patients with cerebovascular disease treated by Rusk, of an average age of 60, 900 were taught to walk and care for themselves and 400 returned to gainful work.

CASE REPORTS

The following are five brief case reports to exemplify the author's experience with anticoagulants on a prolonged basis in patients with cerebrovascular disease:

(1)

Patient: Mr. D. R., Age 74

Seen: Feb. 1, 1954

CC: Obvious weakness in the right hand for the past two weeks. Patient first noticed a sudden onset of dizziness, weakness, awkardness, and numbness of the right hand two weeks previously, while trying to hold some cards in a card game. This condition persisted, and in addition, the patient's wife was aware of some loss of mental acuity over the past year.

On examination, patient's fundi revealed a two plus arteriosclerotic change. His blood pressure was 155/105. Neurological examination revealed that the right arm was ataxic and there was some hyporeflexia. Deep and superficial reflexes were essentially with normal limits, but the point to point and rapid rhythmic alternating movements of the right arm were impaired. The sensory tests were within normal limits.

Patient was started on anticoagulant therapy on 2-19-54, and on 3-1-55 the ataxia had completely subsided and the point to point and rapid rhythmic alternating movements of the right arm were within normal limits. The patient's wife stated that the patient's mental status had improved considerably. The patient has remained on the anticoagulant therapy until the present time (2-4-56) and no further episodes have occurred.

DIAGNOSIS: Insufficiency of the Basilar Artery System.

(2)

Patient: Mr. S. P.

Seen: February 20, 1954

Patient is a white male, age 61, with gangrene of the right leg. He had suffered a coronary thrombosis, myocardial infarction in 1949, and a cerebral vascular accident with a right hemiparesis in 1952.

On examination, the patient had obvious arterial occlusion of the right lower extremity, right hemiparesis, and his speech was thick and slurred, and he was confused.

The electrocardiogram revealed evidence of an old posterior wall infarction, left ventricular hypertrophy, and an occasional premature ventricular systole.

The patient was placed on anticoagulant therapy, together with medical vaso-dilating procedures and he made a slow, steady, and uneventful recovery, and there have been no further thrombo-embolic episodes for the following two years.

DIAGNOSIS: Arteriosclerotic heart disease, with myocardial infarction, mural thrombus and emboli to the leg and to the brain.

(3)

Patient: Mrs. A. S.

Seen: September 10, 1947

CC:

Weakness in the right arm and leg for the previous two weeks. Patient is age 55.

Examination revealed hyperactive deep reflexes in the right arm and leg and a positive plantar response. Patient's blood pressure was 180/130.

Patient was started on anti-hypertensive regime, and anticoagulants were instituted. In June of 1948 the patient discontinued the anticoagulant therapy on her own responsibility, and seven months later suffered a coronary thrombosis and myocardial infarction. Anticoagulants were restarted and the patient has remained on anticoagulants to the present date – a total of 7 years – without further thrombo-embolic episodes.

DIAGNOSIS: Cerebral thrombosis; Coronary Thrombosis with myocardial infarction; Essential hypertension.

(4)

Patient: Mr. C. E. W., Age 57

Seen: April 17, 1946

Patient has a previous history of coronary occlusion and myocardial infarction in August of 1945. This patient was followed on conservative treatment, without untoward episodes until January, 1954, when it was noted that early Parkinson's signs were present. Treatment was instituted with anti-Parkinson's therapy, and the patient improved for a while; however, by November, 1954 there was progressive increase in Parkinson's symptoms and he then developed a momentary loss of reality and felt faint. An electrocardiogram did not reveal changes of significance, but there was rapid loss of mental acuity and increased confusion. It was felt that the patient was having multiple small foci of cerebral thromboses, and anticoagulant therapy was started in December of 1954. Since that time the patient has improved steadily and mental acuity has returned, and there have been no further thrombo-embolic episodes.

DIAGNOSIS: Focal cerebral thromboses;

Parkinsonism; Coronary occlusion and myocardial infarction.

(5)

Patient: Mrs. N. A., Age: 84

Seen: December 24, 1955

Patient has a history of known heart disease, with total irregularity, for the previous five years, with sudden loss of circulation in the left leg, with an area of gangrene on the left heel five weeks before, and then a sudden onset of right hemiparesis and right facial weakness, coma, and aphasia.

Examination revealed an acutely ill woman with auricular fibrillation, coma, and right hemiparesis, and complicating bronchial pneumonia. Patient was started on antibiotics with supporting therapy and anticoagulants. There has been steady improvement. The aphasia has cleared, there are no sensory defects and the remaining problem is the right hemiparesis. Patient is being maintained on anticoagulants.

DIAGNOSIS: Arteriosclerotic heart disease, with auricular fibrillation and multiple emboli to the arm, leg and brain.

CONCLUSIONS

1. The cerebro-vascular diseases today number almost two million victims in the United States and claim an expensive share of the Nation's pocketbook.

2. There is no conformity of terminology and the confusion of words causes a confusion of thought and communication as well as diagnosis and treatment. There is a need for a definition of terms of specific pathological entities.

3. Diagnoses can often be specifically differentiated by detailed physical examinations and appropriate laboratory and X-ray studies.

4. Cerebral blood flow can be improved by appropriate therapeutic measures.

5. Cerebral blood flow can be impaired by inappropriate doses or types of medications.

6. Anticoagulant therapy of thrombo-embolic cerebro-vascular disease has assumed a major therapeutic role in properly selected cases.

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THE MEDICAL MANAGEMENT OF THE ACUTE CARDIAC EMERGENCIES

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FEW conditions tax the physician's diagnostic and therapeutic skill as do acute cardiac emergencies. It is here that lives may be saved by prompt and proper handling of these patients.

Acute Pulmonary Edema is probably the most frequent cardiac emergency for which the physician is called in the early hours of the morning. The patient presents an alarming picture of orthopnea, cyanosis, copious frothy sputum, coarse wet rales, and rhonchi which are heard at a distance. This condition may mask an acute myocardial or pulmonary infarction, and may be the only presenting manifestation of one of these conditions. Failure of the patient to respond to measures about to be detailed should arouse the suspicion of the presence of one of these processes, thereby preventing treatment from being effective.

Treatment:

(1) The patient should be seated upright.

(2) If bronchial asthma or severe pulmonary insufficiency are not present, a hypodermic injection of Morphine Sulphate 10 - 15 mg. (gr. 1/6 - ¼) and Atropine Sulphate 0.5 - 1 mg. (gr. 1/100 - 1/50) is given.

(3) "Bloodless venesection," consisting of blood pressure cuffs placed around both thighs and arms and inflated to the diastolic pressure, is helpful. As the patient improves, the cuffs are released in rotation.

(4) If this is ineffective, a phlebotomy may be done, if anemia is not present.

(5) Aminophyllin 0.5 gm. (gr. 7.5) in 20 cc. of water can be given slowly intravenously.

(6) Oxygen therapy is helpful. A flow of 6 - 7 liters of oxygen per minute should be maintained. 95% of ethyl alcohol in the oxygen humidifying apparatus, used for nasal catheter inhalation, or 30 - 40% ethyl alcohol in the humidifier for the mask decreases intra-alveolar exudation, and so improves ventilation.

(7) Intermittent positive pressure breathing, if available, has been found very effective, using 15 - 20 cm. of water pressure.

(8) If these measures fail, a rapidly acting Digitalis Glycoside should be given. Any one of the following may be given intravenously: Oubain 0.5 mg.; Digoxin 1 mg.; or Cedilanid 0.8 mg.

(9) In refractory acute pulmonary edema associated with hypertension, Arfonad, a ganglionic blocking agent is worthy of trial. The contents of a 5 cc. ampoule (1 cc. contains 50 mg.) are put in 250 cc. of 5% glucose in water and infused at a rate of 10 drops (0.5 mg.) per minute. The rate of flow is gradually increased until a 25 - 30% reduction in the blood pressure level is obtained. The blood pressure is maintained at this level until patient is out of his attack.

Acute Myocardial Infarction: It is beyond the scope of this paper to delve into the diagnosis of this condition or to consider treatment after the acute phase has passed. When first seen at home, the patient is given Morphine gr. 1/4 subcutaneously. If in severe pain, a slow intravenous of Morphine, gr. 1/8 - gr. 1/6, instead, may be given. If ineffective, one-half of the previous dose can be repeated after 5 minutes. Whenever possible, hospitalization is indicated. While awaiting the arrival of the ambulance, Vasoxyl 20 mg. should be given intravenously to maintain the blood pressure. While in the ambulance, the patient should be given oxygen therapy. Upon arrival at the hospital, if the systolic blood pressure is below 90 mm. of mercury, shock therapy should be started at once. The drug of choice is Levophed by intravenous drip. While waiting for this solution to be set up, Vasoxyl 10 mg. is again given intravenously so as not to lose valuable time. Two ampoules (4 cc. per ampoule containing 4 mg.) of Levophed as well as an ampoule containing Heparin Sodium 50 mg. are added to 500 cc. of 5% glucose in distilled water, and infused intravenously at a rate of about 20 drops per minute. The rate of infusion is adjusted so as to maintain a low normal pressure. When the pressure has stabilized at the desired level, the infusion is gradually discontinued over a period of 4 - 8 hours.

Stokes-Adams Attacks refer to attacks of syncope with or without convulsive seizures due to prolonged asytole (5 - 10 seconds or more). The patient loses consciousness and is found to be pulseless. These attacks occur in complete

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heart block, in periods of ventricular tachycardia or fibrillation with or without associated complete heart block.

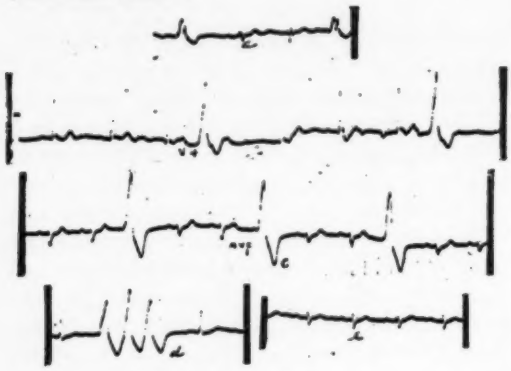

Fig. 1. Stokes-Adams Attack
a. Day before the attack
b. Immediately after the attack
c. 6 days after the attack
d. 12 days after the attack
e. Sinus rhythm following 0.6 gm. Quinidine Sulphate intravenously

Treatment: The drugs of choice are:

(1) Epinephrine 0.3 to 0.5 cc. of 1:1000 dilution subcutaneously for acute episodes of ventricular standstill.

(2) Isuprel 0.2 mg. subcutaneously when the underlying mechanism of the attacks is unknown. If urgency dictates, Isuprel 0.02 mg. may be given intravenously.

Recently the use of an externally applied electrical stimulus, called the Cardiac Pacemaker, has been found to be effective in patients with ventricular standstill who are refractory to other forms of treatment. This method is ineffective in those attacks due to paroxysmal ventricular tachycardia or ventricular extrasystoles.

Paroxysmal Cardiac Arrhythmias arising in individuals with pre-existing organic heart disease, if not checked, will lead to congestive failure, syncopal attacks, embolism, or pulmonary edema. These are supraventricular nodal tachycardia, and ventricular tachycardia. Atrial and/or nodal tachycardia will be referred to as atrial tachycardia for the sake of simplicity, since the therapy for these is identical. An immediate effort should be made to interrupt the arrhythmia.

Paroxysmal Atrial Tachycardia occurs most often in young adults with normal hearts. Its onset is sudden, and the rhythm is regular at a rapid and constant rate of about 160. An important feature of this tachycardia is the constancy of the apical rate. Arising in the course of pre-existing heart disease, especially associated with congestive heart failure, or during a surgical operation, it may constitute a real threat to the patient. When carotid sinus or ocular pressure is effective, this type of tachycardia ends immediately. No other tachycardia responds so dramatically to carotid sinus pressure, but, unfortunately, many paroxysms of atrial tachycardia do not show this response.

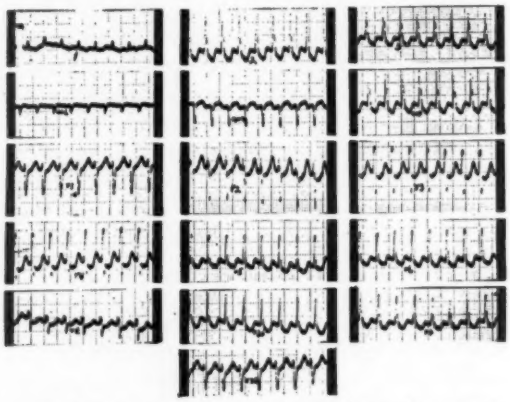

Fig. 2. Paroxysmal Atrial Tachycardia, Rate 214

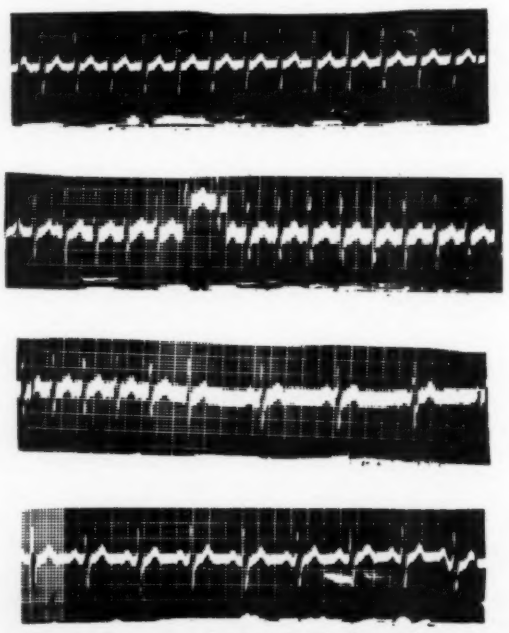

Fig. 3. Paroxysmal Atrial Tachycardia, rate

136, with resumption of rhythm to normal sinus rhythm, rate 93, following carotid sinus pressure.

Treatment: Any one of the vagal maneuvers may be tried:

(1) Carotid sinus pressure;
(2) Ocular pressure;
(3) Valsalva's experiment;
(4) Induction of vomiting either mechanically, or by drugs such as Ipepac or dilute solution of mustard.

If any of these measures is ineffective, a resort to drug therapy is indicated. The following therapy may be mentioned:

(1) Cedilanid, 0.8 mg., given slowly, intravenously, provided the patient has not received any digitalis in the preceding two weeks, is usually effective.

(2) 10% Solution Calcium Gluconate, 10 cc., intravenously.

(3) 20% Solution Magnesium Sulphate, 10 cc., intravenously.

(4) Neosynephrin, 0.5 - 1 mg., intravenously. This drug has stopped attacks in 1 - 2 minutes, but it should be avoided in hypertension or coronary artery disease.

(5) Mecholyl, 25-50 mg., subcutaneously. I caution against the use of this drug because of the alarming period of asystole which may occur before the normal pacemaker resumes control of the rhythm.

(6) Prostigmin Methylsulphate 1 - 2 cc. of a 1:2000 dilution intramuscularly.

While the drugs just listed may not always be effective in restoring normal sinus rhythm, they nevertheless increase the sensitivity of the carotid sinus about 20 minutes after administration, and thereby render the sinus responsive to pressure.

Quinidine has had wide usage in the treatment of paroxysmal atrial tachycardia. The dosage employed is the same as will be described under the management of paroxysmal ventricular tachycardia.

Paroxysmal Ventricular Tachycardia is a serious type of arrhythmia. Generally, a series of six or more ventricular extrasystoles occurring in succession constitute a paroxysm of ventricular tachycardia. It usually occurs in patients with serious coronary artery disease, or digitalis poisoning. The diagnosis of this tachycardia is difficult to establish clinically, and requires

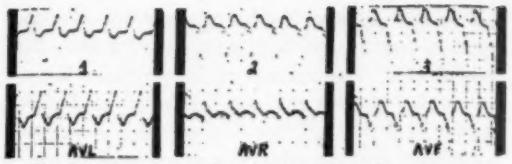

Fig. 4. Paroxysmal Ventricular Tachycardia, rate 150

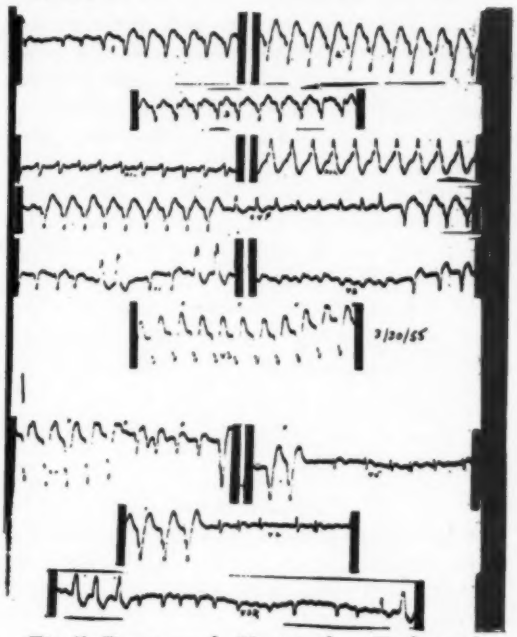

Fig. 5. Paroxysmal Ventricular Tachycardia, rate 166, partially interrupted by 0.6 gm. Quinidine Lactate intravenously.

electrocardiographic means for definite diagnosis.

Treatment: Carotid sinus pressure is ineffective in interrupting a paroxysm. Treatment will depend on the cause. If due to digitalis poisoning, temporarily stopping digitalis and administering Potassium Chloride 1 gram orally three times daily is usually effective. If urgent, Potassium Chloride 0.75 gm. in 100 - 300 cc. of 5% glucose in distilled water can be given intravenously, monitered by frequent serial electrocardiograms.

When this condition is not due to digitalis intoxication, Quinidine or Procaine Amide (Pronestyl) may be used. The route of administration will depend on the urgency of the situation. If paroxysmal ventricular tachycardia occurs during surgery or immediately postoperatively, or when the condition precludes the

oral route and the condition is urgent, the contents of an ampoule of Quinidine Lactate, Hydrochloride, Gluconate, or Sulphate, 0.6 gms. are added to 200 cc. or 5% glucose in distilled water and infused slowly by drip, monitered by direct reading electrocardiographic tracing. The intravenous route is discontinued as soon as reversion to normal sinus rhythm has occurred. When this has occurred, the patient is maintained on Quinidine 0.2 - 0.4 mgs. intramuscularly 3 or 4 times daily until the oral route becomes possible. When the situation is not urgent, the oral route may be used. Quinidine Sulphate 0.2 gm. is given as a test dose. If, after 4 hours, no sensitivity or reactions to the drug occur, 0.6 gm. may be given 4 times a day. The next day, if reversion to sinus rhythm has not taken place, the dose is increased gradually to 1 gram 4 times a day. It is important to maintain strict electrocardiographic control once the heart has slowed.

Fig. 6. Paroxysmal Ventricular Tachycardia, rate 150, with conversion to sinus tachycardia, rate 120, following intravenous administration of 100 mg. Pronestyl.

The mistake often made in Quinidine therapy is that the patient is not given enough of this drug. Occasionally, when reversion has not occurred, a subcutaneous or intravenous injection of Atropine Sulphate 1 - 2 mg. (gr. 1/60 - 1/30) given one-half hour after the last and largest dose of Quinidine may restore the heart to normal rhythm.

Procaine Amide (Pronestyl) may be given orally in doses of 0.25 - 0.5 gm. every 2 - 4 hours until the attack is controlled. When urgency dictates, the contents of a 10 cc. ampoule (1 cc. contains 100 mg.) of Procaine Amide (Pronestyl) are injected intravenously no faster than 1 cc. per minute, monitered by a direct reading continuous electrocardiographic tracing.

Atrial Fibrillation and Atrial Flutter will be considered together because the treatment is similar in both.

Atrial Fibrillation is almost always observed in myocardial disease of an advanced grade. It is observed in 60% of patients with heart failure. Frequently associated factors are hypertensive, arteriosclerotic, cardiovascular disease, rheumatic heart disease, thyrotoxicosis and toxic states. Auscultation reveals a totally irregular rhythm. If apical and radial rates are compared simultaneously, a pulse deficit is noted. Multiple extrasystoles at times are difficult to differentiate from atrial fibrillation. Exercise usually diminishes the extrasystoles, but increases the irregularity of atrial fibrillation. Here again, the electrocardiogram is most helpful.

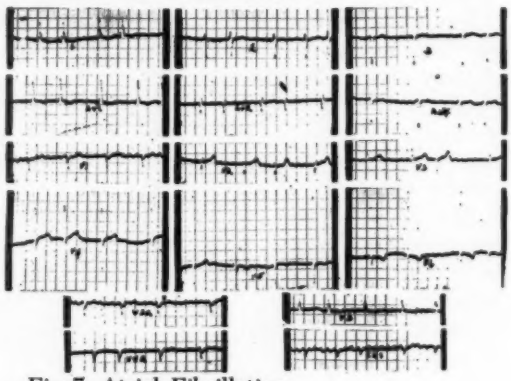

Fig. 7. Atrial Fibrillation

Atrial Flutter is usually seen in patients with moderately advanced or severe myocardial disease. The symptoms of this condition are similar to those of other types of rapid heart action except for a greater tendency to develop heart failure. The diagnosis is established by the electrocardiogram.

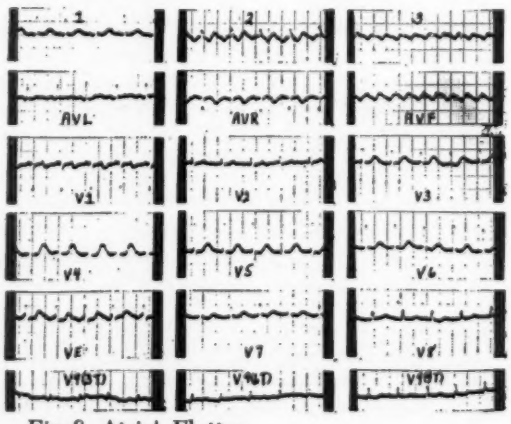

Fig. 8. Atrial Flutter

Treatment: Digitalis is the drug of choice. Digitalization should be performed rapidly if a severe grade of heart failure is present and/or when the rate is rapid. One of the following

should be given intravenously, followed in four hours by a similar dose:

Cedilanid 4 cc. (0.8 mg.) or

Digoxin 3 cc. (.75 mg.)

This should be followed by daily oral maintenance doses of either Gitaligin 0.5 or Digoxin 0.5 mg. The conversion of this arrhythmia is beyond the scope of this paper.

Ventricular Fibrillation is usually viewed as a terminal and terminating incident in certain instances of sudden death; such as, during an attack of angina pectoris or acute coronary occlusion, and following anesthesia. It is an extreme arrhythmia characterized by rapid, irregular, uncoordinated and ineffective twitchings of the ventricle. The electrocardiogram shows rapid regular or irregular oscillations representing bizarre QRS complexes. Quinidine administered intravenously have been found useful in those cases which have survived long enough to receive this medication. The dosage is the same as that given for paroxysmal ventricular tachycardia.

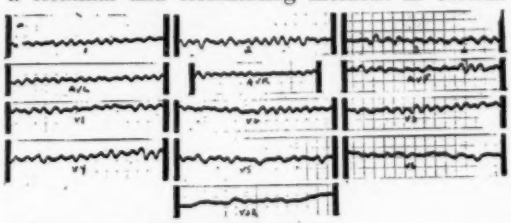

Fig. 9. Ventricular Fibrillation

Summary:

1. The commonly occurring acute cardiac emergencies have been presented.

2. Treatment for these conditions was outlined.

PHOENIX *Clinical* CLUB

The Case History in this discussion is selected from the Case Records of the Massachusetts General Hospital, and reprinted from the New England Journal of Medicine. The discussant under Differential Diagnosis is a member of the staff of the Massachusetts General Hospital. The other discussants are members of the Phoenix Clinical Club.

MASSACHUSETTS GENERAL HOSPITAL
PRESENTATION OF CASE

A FORTY-THREE-YEAR-OLD woman was admitted to the hospital with dysphagia and headache.

The patient was in apparent good health until two and a half months prior to admission, when she noted difficulty in swallowing liquids. Attempts to drink milk, for example, were followed by choking and she had difficulty in getting her breath. The dysphagia was not always present, and she apparently had no difficulty in swallowing solid foods. She also noted that her voice was weak and hoarse. Lying down at night brought on a feeling of oppression in the chest, and was not relieved by sitting up. A sense of obstruction was present in the throat resulting in constant unproductive attempts to clear the throat. There was no caughing or expectoration. Three weeks before entry she developed severe pounding headache aggravated by motion. It was worse in the morning, often beginning as a generalized throbbing and finally localizing in the right temple. Two weeks prior to admission she noted marked weakness of the right arm and was unable to control its movement when reaching for things. The legs felt stiff, and she seemed unable to lift them. Sharp pains were present in the left leg. About four months before admission the patient had several episodes of pain and fullness in the epigastrium that came on about an hour after meals. The The distress was relieved to some extent by a hot-water bottle but not by food. A local physician made the diagnosis of gastric ulcer by fluoroscopy. Following treatment the symptoms disappeared and at the time of admission she had been free of epigastric distress for three months.

Physical examination revealed a poorly developed, emaciated and dehydrated woman with a weak, hoarse voice. The breasts were poorly developed but otherwise negative. There was no adenopathy. The pupil reacted to light and accommodation. The gait was shuffling. The Romberg sign was positive. The right shoulder drooped and could not be raised; the muscles of the right shoulder and upper arm showed

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dry-filled sealed capsules

[1]Albertson, H.A. and Trout, H. H., Jr.: *Antibiotics Annual* 1954-55, Medical Encyclopedia, Inc., New York, N.Y., 1955, pp. 599-602.

[2]Prigot, A.; Whitaker, J. C.; Shidlovsky, B. A., and Marmell, M.: *ibid*, pp. 603-607.

Lederle

LEDERLE LABORATORIES DIVISION
AMERICAN CYANAMID COMPANY
PEARL RIVER, NEW YORK

*REG. U.S. PAT. OFF.

PHOTO DATA: AERIAL CAMERA WITH K-2 FILTER AT DUSK, F.11, 4/100 SEC., FAST PAN FILM

ACHROMYCIN ACHROMYCIN

hypotonicity and some atrophy. There was a tendency toward "winging" of the right scapula. All the deep tendon reflexes were hyperactive, more so on the right. The right lower abdominal reflex was diminished. The plantar responses were flexor in type bilaterally. The finger-to-nose and heel-to-knee tests showed awkwardness and overshooting bilaterally, but more so on the right.

The temperature was 98.6°F., the pulse 94, and the respirations 20. The blood pressure was 116 systolic, 70 diastolic.

Examination of the blood showed a red-cell count of 4,520,000, with 14 gm. of hemoglobin, and a white-cell count of 8500, with 60 per cent neutrophils. The urine and stools were negative. The serum protein was 6 gm. per 100 cc.; the blood sugar and the serum nonprotein nitrogen were normal. A tuberculin test in a dilution of 1:1000 was negative. A Hinton test was negative.

A roentgenogram of the chest revealed a few linear shadows in t left third and fourth interspaces anteriorly. An ill-defined shadow was present in the region of the left hilus. An x-ray examination of the skull was negative. Two electroencephalograms taken soon after admission revealed a diffuse spotty dysrhythmia without focal distribution but generally worse toward the occiput.

A lumbar puncture revealed clear colorless fluid. The pressure was normal initially and responded normally after jugular compression. No cells were present; the protein content was normal, and the Wassermann test was negative. An examination of the larynx revealed complete paralysis of the left vocal cord, which lay in the so-called "cadaveric position." The right cord functioned normally.

On the twentieth hospital day the temperature gradually began to rise, reaching 102°F. over a period of about thirty-six hours. She developed some cough, raising a small amount of sputum, and there was pain over the left anterior chest on coughing. The left apex and upper chest were dull to percussion, with bronchial breath sounds anteriorly and an expiratory grunt. There were coarse rales over the left apex posteriorly. The liver was palpable two fingerbreadths below the costal margin. A sputum culture revealed alpha-hemolytic and a few beta-hemolytic streptococci, and a blood culture was negative. The white-cell count was 24,000. A catheterized urine specimen was negative. A roentgenogram of the chest revealed irregular patchy consolidation of the left upper lobe and a small amount of fluid in the posterior costophrenic sinus.

The temperature continued to range between 100 and 102.6°F. Respirations were shallow and rapid, and the patient appeared deyhdrated and slightly cyanotic. She was treated with sulfadiazine and oxygen. On the twenty-sixth hospital day, the patient became jaundice and a moderate amount of bile appeared in the urine. Sulfadiazine was stopped and penicillin was substituted. She was also given potassium iodide because of difficulty in bringing up the tenacious sputum. Diffuse coarse rales were audible throughout the lung field.

On the thirtieth hospital day the patient began to improve. The temperature gradually fell to normal and the sputum diminished in quantity. The icterus cleared completely, and the liver was no longer palpable. The lungs showed slight dullness over the entire left chest with scattered fine and coarse rales, and normal breath sounds diminished over the left apex anteriorly. There was slight sacral edema. The total plasma protein was 5.3 gm. per 100 cc., with an albuminglobulin ratio of 0.93. The serum chloride was 90 milliequiv. per liter. A cephalin flocculation test was negative in twenty-four and in forty-eight hours. X-ray examination of the chest revealed little change in the appearance of the left upper lobe.

The temperature continued to range between normal and 100°F., occasionally reaching 101°F. The neurologic signs originally noted became more marked, with extreme weakness of the right arm. A positive Hoffman sign appeared on the right, but the Babinski signs were negative. Dulness and bronchial breath sounds reappeared over the left upper lobe and were accompanied by scattered fine rales and a cough, which was moderately productive of purulent sputum. Another chest film revealed no change in the left upper lobe, but the right lung field showed increased linear markings. Pleural fluid was present bilaterally. Several convulsions of the Jacksonian type occurred, each lasting several minutes. The convulsive movements started in the right hand and arm, spreading to the leg and face. The seizures

were followed by drowsiness and sleep. On the fortieth hospital day a Babinski sign was elicited on the right and there was prolonged ankle clonus on the right. There was dullness to pinprick in the right leg. On the same day the patient had a convulsion followed by temporary aphasia.

She continued to cough and raise large quanities of green mucoid sputum. She became uncooperative, refusing all food and medication. The chest signs remained unchanged. She became extremely weak and drowsy and expired on the fifty-third hospital day following a convulsive episode.

DR. JOSEPH MADISON GREER

A 43 year old woman with dysphagia and headaches for two and a half months. Difficulty in swallowing liquids, was hoarse and had headaches in the right temple. Oppression in the throat, (could swallow solids but not liquids). The headache was aggravated by motion localized in the right temple. Weakness and lack of control of the right arm; shoulder drooping, legs stiff and heavy.

Past history unimportant but her doctors diagnosed gastric ulcer and cured her before she came into the hospital.

She developed what was probably pneumonia while she was in the hospital and this subsided under treatment. She had convultions and these increased in frequency and she went right on and died on the 53rd hospital day in spite of all they could do.

I will review some of the important points of the Physical Examination for my own thinking as well as for some of the fellows who perhaps have not read the case (I know we are not supposed to do this but just for fun how many HAVE read the case?)

She was poorly nourished, had a weak hoarse voice, a shufflings-gait with a positive Romberg and the co-ordination was interfered with which to me indicates something wrong with the cerebellum. The right shoulder drooped and could not be raised; This suggests some involvement of the 11 cranial nerve the spinal accessory and the circumflex and these have their origin in the 5th & 6th cervical. The deep tendon reflexes were hyperactive which indicates a lesion in the upper motor neuron.

At first the blood picture was not remarkable and there was no temperature. (This picture was changed during the time of her stay in the hospital.) However, at the time of entrance into the hospital there was something in the chest and there was an indefined shadow in the region of the left hilus. It seems to me that this is significant. We should remember this in our discussion.

I do not know too much about Electroencephalograms but it was stated that it was worse toward the occiput which is in the region of the cerebellum.

We are told that there was complete paralysis of the left vocal cord. This would account for her hoarse voice and an associated condition from probably the same origin would explain why she could not swallow and the fact that she could swallow solids and not liquids would rule out organic constricion and indicate spasm which would mean that it was of nerve origin. Was this a central or high cord lesion or a peripheral lesion. I can find no suggestion that it could be peripheral in the protocol so I shall assume that it was central. The recurrent laryngeal nerve supplies the vocal cords and is a branch of the Vagus or 10 cranial nerve. This gets back to the neighborhood of the base of the brain and we now note that we have a paralysis of the left vocal cord and a weakness of the right arm. As we remember these tracts crossover someplace in this area.

We now are told that she has a positive Hoffman sign (which is a flexion of the terminal phalanx of the thumb when we click the end of the index finger from flexion to extension and is said to indicate some involvement of the lateral motor tract.) The Babinski sign was negative at first, but appeared later.

Epileptiform convultions appeared, of the Jacksonian type, and started in the right hand and arm. This usually means an organic lesion in the motor cortex, but may be more definitely localized.

At first there was no temperature and the blood was essentially normal but on the 20th hospital day she began to have temperatures up to 102 and a blood count of 24,000 and there was something more in the chest. She developed cough, sputum and pain over the left chest and the left apex was dull and the x-ray showed patchy consolidation and some fluid. This was no doubt a pneumonitis and

about the same time she developed an acute hepatitis and she had a large and tender liver and became jaundiced. This was the 26th hospital day.

On the 30th hospital day she began to improve and the jaundice cleared completely. They had cured her pneumonia and hepatitis with sulfadiazine and penicillin. The blood chemistry was relatively normal but the x-ray examination revealed little change in the appearance of the left lobe of the lung. The patient was not cured and continued to have temperature although not so high and the neurologic signs became more marked.

On the 40th day the chest findings were increased and she had convulsions and a Babinski sign was noted on the right as well as angle colonus and sensory changes in the right leg. The patient continued to have convulsions and developed aphasia. There was continued cough and sputum and she refused all food and medication, became uncooperative and expired on the 53rd hospital day, following a severe convulsion.

DISCUSSION:

The neurological signs and symptoms seem to be the most important and indicate a lesion in the brain.

Is this a vascular pathology? I would say not. It could hardly be luetic as both blood and spinal fluid were negative. Could it be a vascular accident? I would say not as she is not in the age group nor is there any indication of circulatory trouble, that would indicate a hemorrhage or an embolus or thrombus. Could it be an infectious process with brain abscess or meningitis? Yes I suppose it could be and sometimes brain abscesses are hard to diagnose. However, she had trouble before we had any history of infection.

Could this lesion be in the nature of a new growth? Yes, I think that it could. If so is it primary in the brain or is it metastatic? If it is metastatic where is the original tumor? We have been told at two different times about the lesion in the left lung and I do not think that this can be ignored.

Therefore, it is my opinion that the condition is a metastatic new growth and that the original tumor is a tumor in the hilus or bronchus of the left lung. Probably a bronchogenic carcinoma. She could also have had metastasis to the liver.

DIFFERENTIAL DIAGNOSIS

DR. ARTHUR LINENTHAL: The problem in this patient appears to be one of disease involving structures within the chest as well as the central nervous system. Consideration of the neurologic lesion may appropriately follow discussion of the chest difficulty, since I think that the two are interrelated.

The presenting symptoms of this patient were difficulty in swallowing, difficulty in breathing, a sense of opression in the chest, a sense of obstruction in the throat, and weakness and hoarseness of the voice. The complete paralysis of the left vocal cord, fixed in the cadaveric position midway between phonation and quiet inspiration, and the ill-defined X-Ray shadow in the left hilar region suggested that these findings as well as the symptoms may have been due to some process in the left hilar region involving the left recurrent laryngeal nerve and pressing on the esophagus and trachea. The emaciated condition of the patient on admission is consistent with the impression that a malignant growth was the likeliest basis of such a process.

Regarding the primary site of such a tumor, bronchogenic carcinoma seems most probable, although other locations must be considered. It is interesting that there had been no cough and no sputum before the patient entered the hospital, but this does not rule out a pulmonary neoplasm.

The statement that a gastric ulcer had been seen by fluoroscopy raises the question whether there was a gastric neoplasm with metastases to the hilar nodes and surrounding structures. Since the abdominal symptoms that led to the fluoroscopy subsided, X-ray studies of the stomach were not repeated, and we have no evidence on which to settle this question.

There is no evidence for other diseases that might have given rise to the presenting symptoms and physical findings. Neither physical examination nor X-ray examination showed evidence of an aortic aneurysm, and examination of the larynx did not reveal any intrinsic disease. Esophageal carcinoma might well have explained the presenting picture, but since no studies of the esophagus were made, there is no way to confirm this diagnosis.

At the end of the third week in the hospital, the patient developed a pulmonary infection in

the left upper lobe. This was marked by fever, cough, sputum and pain over the left chest. The respirations were rapid and shallow, and she became cyanotic. Physical examination gave evidence of consolidation over the left upper lobe, and this was confirmed by X-ray. Elevation of the white cell count suggests a bacterial etiology, but the sputum showed only alpha-hemolytic and a few beta-hemolytic streptococci. She was given sulfadiazine and then penicillin, but there was no evidence that these were of value. The patient improved, the physical findings over the left lobe diminished but never entirely disappeared, and the X-ray picture showed little change.

It is interesting that this severe pulmonary infection developed in the left upper lobe, since the lesion that I have supposed to exist at the left hilus could easily have compressed the upper lobe bronchus and caused some increased susceptibility to infection in that area. The linear shadows seen in the third and fourth left interspaces on the first chest film may have represented small areas of atelectasis.

At the time of the development of the pulmonary infection, before the patient had received any chemotherapy, the liver was found to be enlarged. Subsequently she was given sulfadiazine, and while receiving the drug, she became jaundiced and had bile in the urine. The sulfadiazine was stopped and soon afterward, along with improvement in the pulmonary condition, the jaundice disappeared and the liver became smaller.

It is difficult to be sure of the relation between the pulmonary infection, the sulfonamide therapy and what appears to have been an episode of acute hepatitis. Toxic hepatitis has been described after sulfadiazine, but the presence of the hepatic enlargement in this case before the sulfadiazin was given makes me believe that the sulfa drug was probably not responsible. Bacterial pneumonia may be accompanied by jaundice. There is also the possibility that both the pulmonary infection and the hepatitis were due to a virus infection. The negative cephalin flocculation test done at about the time the jaundice was subsiding suggests that the degree of liver damage was slight. No other liver function tests were done. The plasma proteins showed no significant change.

Obstructing lesions of the biliary tract must be mentioned, but there is nothing to support such a diagnosis and the transient hepatic enlargement is suggestive of intrahepatic disease. There was apparently no abdominal pain at that time, and there is no evidence of gallstones. Metastatic disease of the liver or around the bile passages seems too remote to be seriously considered.

The pulmonary infection never entirely cleared up, and indeed at the time of death there was an increase in the physical findings, as well as a persistence of the X-ray abnormalities over the left upper lobe and the development of purulent sputum. Apparently the infection was a persistent one, either because of some blockage of drainage from the involved lobe or because of the characteristics of the infectious process. This may well have been a virus disease with superimposed bacterial infection.

The acuteness of the onset of the pulmonary infection, together with the absence of clinical or X-ray evidence of pulmonary tuberculosis on admission, seems to eliminate tuberculosis as a possibility. Unfortunately, there is no record of search for tubercle bacilli in the sputum.

The neurologic disorder was, I think, related to the disease in the chest; that is, it was caused by a metastasis of the malignant process. The problem here is to decide on the basis of the evidence presented how many neurologic lesions were present and where they were located.

The terminal neurologic findings were marked. The weakness in the arm, the appearance of a Hoffman sign, the development of Jacksonian convulsions starting in the right arm and hand, the appearance of a Babinski sign on the right with ankle clonus, the increase in the deep tendon reflexes on the right and the episode of temporary aphasia point to a lesion in the left cerebral hemisphere involving primarily the motor area for the arm with extension to the motor area for the leg and to the nearby speech area. Sensory changes, such as the late dullness to pinprick in the right leg, suggests subcortical extension of the process.

The earliest manifestation of any neurologic disorder was the development of weakness in the right arm. Subsequently the muscles of the right arm and the right shoulder girdle showed atrophy. This finding suggests a metastic lesion involving the anterior-horn cells

of the right side of the mid-cervical spinal cord. There was hypotonicity, but the deep tendon reflexes were also increased. The atrophy can be explained on the basis of weakness of the right arm with resulting disuse. The stiffness of both legs at the onset is difficult to explain. The positive Romberg sign and the evidence of incorrdination on admission may be attributed to weakness. It seems, therefore, that all the findings can be explained on the basis of one metastatic cerebral lesion.

The patient went down hill rapidly and died following a convulsive seizure, probably owing to involvement of a vital nervous center.

CLINICAL DIAGNOSIS

Tumor involving left motor cortex or brain (? primary, ? metastatic from bronchiogenic carcinoma).

Unresolved pneumonia: left upper lobe.

DR. LINENTHAL'S DIAGNOSIS

Bronchiogenic carcinoma of the left lung, with metastases to regional nodes and left cortical and subcortical regions of brain.

Unresolved pneumonia: left upper lobe.

Acute hepatitis, subsiding.

ANATOMICAL DIAGNOSIS

Carcinoma, oat-cell type, of left upper lobe, with metastases to bronchial, mediastinal, mesenteric and retroperitoneal lymph nodes, liver, kidney and brain.

Chronic pneumonitis: left upper lobe.

Bronchiectasis: left lower lobe.

Cerebral pressure cones.

PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: As Dr. Linenthal predicted, the pulmonary and cerebral lesions were related. This woman did have a bronchiogenic carcinoma, the primary focus being in the bronchus to the left upper lobe, which was completely occluded. The metastatic bronchial and mediastinal lymph nodes had infiltrated the wall of the left main bronchus, especially the lower lobe bronchus. The latter was so narrow that secondary bronchiectasis had developed through the lower lobe, the bronchioles being dilated and filled with purulent material. The primary tumor in the left upper lobe had extended into the parenchyma around the bronchus for about 4 cm., and the remaining pulmonary tissue in this lobe was gray and rubbery, characteristic of the so-called "drowned out" or "stasis" pneumonitis.

There were three cerebral metastases. The largest, and the one that caused most of the cerebral symptoms, was a systic lesion measuring 6 by 3 by 2.5 cm. in the left parietal region. The other two, each about 2 cm. in diameter, were solid nodules, one in the right posterior frontal region and one in the posteroinferior portion of the right cerebellar hemisphere. There was definite evidence of increased intracranial pressure, as shown by flattening of the convolutions and the deep cerebellar and temporal pressure cones.

The liver weighed over 2000 gm. and contained many metastatic nodules. I do not believe, however, that there was sufficient replacement or parenchyma to have produced even the transient jaundice. A likelier explanation is the presence of many metasatic lymph nodes surrounding and compressing the common bile duct.

Microscopically the caricnoma was extremely undifferentiated, showing no tendency to either keratinization or gland formation. In many places it suggested the oat-cell type.

Editorial

ARIZONA MEDICINE
Journal of
ARIZONA MEDICAL ASSOCIATION, INC.

VOL. 13 JULY, 1956 NO. 7

CONTRIBUTORS

The Editor sincerely solicits contributions of scientific articles for publication in ARIZONA MEDICINE. All such contributions are greatly appreciated. All will be given equal consideration.

Certain general rules must be followed, however, and the Editor therefore respectfully submits the following suggestions to authors and contributors:

1. Follow the general rules of good English, especially with regard to construction, diction, spelling, and punctuation.
2. Be guided by the general rules of medical writing as followed by the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION.
3. Be brief, even while being thorough and complete. Avoid unnecessary words. Try to limit the article to 1500 words.
4. Read and re-read the manuscript several times to correct it, especially for spelling and punctuation.
5. Manuscripts should be typewritten, double spaced, and the original and a carbon copy submitted.
6. Articles for publication should have been read before a controversial body, e.g., a hospital staff meeting, or a county medical society meeting.
7. Exclusive Publication—Articles are accepted for publication on condition that they are contributed solely to this Journal. Ordinarily contributors will be notifed within 60 days if a manuscript is accepted for publication. Every effort will be made to return unused manuscripts.
8. Illustrations — Ordinarily publication of 2 or 3 illustrations accompanying an article will be paid for by Arizona Medicine. Any number beyond this will have to be paid for by the author.
9. Reprints — Reprints must be paid for by the author at established standard rates.

The Editor is always ready, willing, and happy to help in any way possible.

JOINT COMMISSION ON ACCREDITATION OF HOSPITALS

RECENTLY the Joint Commission on Accreditation of Hospitals as made up by the American Medical Association, American Hospital Association, College of Physicians and College of Surgeons has demanded that an organized plan for disaster be available in hospitals before they will be accredited. This is a desirable move for in all plans that ring of "Civil Defense," we, the American Public and Medical Profession move too slowly. As time goes on it becomes imperative that we take out this type of insurance, that plans be formulated and placed in reserve. We strongly commend this action of the Joint Commission on Accreditation of Hospitals.

It is obvious that Phoenix and Tucson are critical target areas with the great majority of our hospital beds and trained personnel in these communities. A great portion of both would be destroyed. The outlying communities would receive casualties and evacuees from these prime target areas. Further, the communities throughout the State must plan to receive casualties from the West Coast if war develops.

Joint planning between the Southwest communities of Albuquerque, Phoenix, El Paso and Tucson would be desirable. This recommendation has been made to Civil Defense authorities a number of times during recent years but no progress has been made for this coordinated effort.

Recently at the Arizona Medical Association Meeting a 200 bed mobile hospital was available. It attracted little interest, which is understandable. However, to maintain this unit available in our State would certainly be a desirable move and Civil Defense authorities should be encouraged to take every step possible to have this added emergency facility available for our relatively isolated segment of the country which we must make as self sufficient as possible.

D. W. N.

EXAMINATIONS FOR FELLOWS OF INTERNATIONAL COLLEGE OF SURGEONS

Examinations for qualified fellows of the International College of Surgeons will be held in Chicago, July 23-24 and October 29-30.

Oral conferences will be held on August 6 and October 22.

For details, write to the Secretary of the Qualifications Council, International College of Surgeons, 1516 Lake Shore Drive, Chicago 10, Illinois.

REPORT OF THE DELEGATES

ACTIONS OF THE HOUSE OF DELEGATES

American Medical Association, 105 Annual Meeting

June 11-15, 1956, Chicago

THIS summary of the recent meeting of the A.M.A. House of Delegates in Chicago is published at this time as an aid to the membership of our State Association in familiarizing each one with actions taken on important topics under discussion. It covers only a few of the many important subjects dealt with by the House, and is not intended as a detailed report on all actions taken.

Your Executive Secretary, Mr. Robert Carpenter and your Delegate attended all the meetings of the House. The Delegate was assigned by the Speaker to the Reference Commitee on Rules and Order of Business.

Our former Governor, Mr. Howard Pyle, appeared on the Program arranged on Sunday afternoon by the Conference of State Presidents and other Officers. He delivered an address which was very well received by a large audience, on the "Body Politic." Mrs. Hamer and I entertained him at supper before he returned to Washington that evening to be near the President of the U. S. who had just undergone surgery.

Dr. David B. Allman, surgeon of Atlantic City, N. J. was named as president-elect for the coming year. A member of the A.M.A. Board of Trustees since 1951, and also chairman of the Committee on Legislation, Dr. Allman will become president of the American Medical Association at the June, 1957 meeting in New York City. He will then succeed Dr. Dwight H. Murray, Napa, Calif. who took office at the Tuesday evening inaugural program in the Chicago Civic Opera House.

Hospital Accreditation

The House of Delegates approved the report of the Committee to Review the Functions of the Joint Commission on Accreditation of Hospitals, which was appointed by the Speaker as a result of action taken at the June, 1955, meeting. The Committee came to the following conclusions:

"1. Accreditation of hospitals should be continued.

"2. The Joint Commission should maintain its present organizational representation.

"3. The Board of Trustees should report annually to the House of Delegates on the activities of the Joint Commission.

"4. Physicians should be on the administrative bodies of hospitals.

"5. General practice sections in hospitals should be encouraged.

"6. Staff meetings required by the Joint Commission are acceptable, but attendance requirements should be set up locally and not by the Commission.

"7. The Joint Commission should not concern itself with the number of hospital staffs to which a physician may belong.

"8. The Joint Commission is not and should not be punitive.

"9. The Joint Commission should publicize the method of appeal to hospitals that fail to receive accreditation.

"10. Reports on surveys should be sent to both administrator and chief of staff of hospital.

"11. Surveyors should be directly employed and supervised by the Joint Commission.

"12. Surveyors should work with both administrator and staff.

"13. New surveyors should receive better indoctrination.

"14. Blue Cross and other associations should be requested not to suspend full benefits to non-accredited hospitals until those so requesting have been inspected.

"15. The American Medical Association should conduct an educational campaign for doctors relative to the functions and operations of the Joint Commission.

"16. The Committee also suggests that the American Medical Association and the American Hospital Association encourage educational meetings for hospital boards of trustees and administrators either on state or national levels to acquaint these bodies with the functions of accreditation.

"17. This Committee asks to be discharged upon submission of this report to the House of Delegates."

The House also approved a reference com-

mittee suggestion that the following statement be added to strengthen the report:

"The Committee recommends that the commissioners to the Joint Commission on Accreditation of Hospitals, appointed by the Board of Trustees of the American Medical Association, urge that Commission to study:

"1. The problems of the exclusion from hospitals and arbitrary limitation of the hospital privileges of the general practitioner, and

"2. Methods whereby the following stated principles may be achieved:

" 'The privileges of each member of the medical staff shall be determined on the basis of professional qualifications and demonstrated ability.'

" 'Personnel of each service or department shall be qualified by training and demonstrated competence, and shall be granted privileges commensurate with their individual abilities.' "

Graduates of Foreign Medical Schools

The House of Delegates approved in principle a program for the evaluation of graduates of foreign medical schools seeking hospital positions in the United States. The proposed program was developed by the Cooperating Committee on Graduates of Foreign Medical Schools, representing the A.M.A. Council on Medical Education and Hospitals, American Hospital Association, Association of American Medical Colleges and Federation of State Medical Boards of the United States.

The following principles were emphasized by the Council on Medical Education and Hospitals in its report recommending A.M.A. participation in the program.

"1. Although the responsibility to share educational opportunities in medicine is recognized, the primary concern must be for the health care of the American public. Thus, before assuming responsibility for the care of patients as interns or residents, all graduates of foreign medical schools (immigrants, exchange students and American graduates of foreign medical schools) should give evidence, as nearly as can be measured, of having reached a level of educational attainment comparable to that of students in American schools at the time of graduation.

"2. The primary objective of this Committee is to devise an effective mechanism for measuring educational attainment in the absence of intimate and continuing knowledge of the educational background of foreign-trained physicians. This mechanism should provide hospitals with pertinent information regarding the medical qualifications of foreign-trained physicians seeking positions as interns or residents. It should not interfere with the hospital's privilege of making its own selection among qualified physicians, nor should it serve as a substitute for or interfere with the normal licensure procedures of the various state boards.

"3. It is not intended that this mechanism be applicable to those foreign medical school graduates in this country as temporary students participating in programs of medical and related studies in recognized universities, medical schools and postgraduate schools, who by the very nature of their study are not involved in the responsibility of patient care."

The proposed plan calls for establishment of a central administrative organization to evaluate the medical credentials of foreign trained physicians desiring to serve as interns or residents in American hospitals. Basic requirements would include satisfactory evidence of at least 18 years of total formal education, including a minimum of 32 months in medicine exclusive of any time which in this country would be considered as premedical study or internship. Applicants with satisfactory credentials then would take a screening examination to determine their medical knowledge and their facility with the English language. Successful applicants then would be certified to hospitals and other interested organizations, with the approval of the foreign-trained physician concerned.

Private Practice by Medical School Faculty Members

Another major action by the House involved the problem of private practice by medical school faculty members, which has been under study by the Committee on Medical and Related Facilities of the Council on Medical Service. The House adopted a Council report which stated "that it shall be the policy of the American Medical Association that funds received from the private practice of medicine by salaried members of the clinical faculty of the medical school or hospital should not accrue to the general budget of the institution and that the initial disposition of fees for medical service from paying patients should be under the direct

control of the doctor or doctors rendering the service."

It was further recommended that adequate liaison be developed and maintained between each county medical society and any medical school or schools in its area; that the Council on Medical Education and Hospitals and the Association of American Medical Colleges urge all medical schools to assist and work with medical societies in developing such liaison, and publicity emanating from a medical school should be in good taste and of a type which has the approval of the general medical community in that area.

The adopted report also said: "It is not in the public or professional interest for a third party to derive a profit from payment received for medical services, nor is it in the public or professional interest for a third party to intervene in the physician-patient relationship."

Federal Aid to Medical Schools

One of the most controversial subjects of debate on the floor of the House was a resolution expressing strong opposition to S. 1323, a bill in Congress providing for one-time, matching grants to medical schools for construction purposes. The Association in recent years has been supporting such legislation in principle, with certain reservations concerning details of some provisions. The House reaffirmed that policy by approving a reference committee statement which said:

"We appreciate the intent with which this resolution was introduced, but at the same time we feel that there are many economic and geographical factors involved, which might not make this resolution practical on a national level. Inasmuch as no evidence was offered to this Committee to justify a change in the previously declared policy of the House of Delegates, your Committee recommends that this resolution be not adopted."

Premature Drug Publicity

The House adopted a substitute resolution which read:

"Whereas, In recent years, events have indicated the necessity for a closer liaison between the pharmaceutical manufacturer and the American Medical Association; and

"Whereas, In view of the tremendous number of new drugs being developed and the expanding research programs in medical colleges, clinics and hospitals being financed by the drug industry, it is imperative that the manufacturer and the medical profession develop cooperatively guiding principles which will protect the American people from being subjected to the premature release of information pertaining to new products or techniques; and

"Whereas, Competition within the pharmaceutical industry has become extremely keen so that in the advertising of their drug products manufacturing firms have been forced into the expenditure of larger and larger sums of money and in increasingly broader fields of advertising; therefore be it.

"Resolved, That the Board of Trustees of the American Medical Association appoint a liaison committee to meet with representatives of the pharmaceutical manufacturers to accomplish this objective."

Miscellaneous Actions

Among many other actions on a wide variety of subjects, the House also:

Approved a Board of Trustees statement on Social Security which included the following: "It is imperative that we distinguish clearly between this problem of coverage of physicians and the far more dangerous disability proposal. The fact should be recognized that the shape of medical practice in the future is not directly related to the inclusion or exclusion of physicians under OASI. It is a matter of vital importance to us as individuals, but it cannot, per se, stimulate further governmental intrusion into medical care. On the other hand, the disability amendment obviously brings the Social Security Administration closer to the regulation of medical care than ever before."

Adopted a resolution amending the By-laws to provide that the Vice President, Treasurer, Speaker and Vice Speaker of the House of Delegates shall be ex officio members of the Board of Trustees with all the rights and duties of the Board without the right to vote.

Increased membership of the Council on Medical Service from six to nine active or service members and eliminated all ex officio members except the immediate Past President.

Directed the Council on Medical Service and the Council on Industrial Health to reconsider the "Guiding Principles for Evaluating Management and Union Health Centers" through their joint Committee on Medical Care for Industrial

Workers and to so revise the guides that they conform completely with the Principles of Medcial Ethics.

Authorized the Committee on Federal Medical Services to make a continuing study of all aspects of VA medical activities under the basic policy established in June, 1953, and suggested reconsideraiton of the temporary exceptions made at that time with respect to neuropsychiatric and tuberculosis disorders.

Recommended that the Board of Trustees select New York City as the place of the 1961 annual meeting.

Medical Ethics

Perhaps one of the most important matters of business considered by the House of Delegates was the proposed NEW PRINCIPLES OF MEDICAL ETHICS. The material was prepared and submitted by the Council on Constitution and Bylaws of the A.M.A. The Council on Constitution and Bylaws had the cooperation and approval of the Judicial Council.

The reference committee approved the material and recommended that the House of Delegates take action at the next session of the House of Delegates.

For your information and consideration, the proposed new condensed PRINCIPLES OF MEDICAL ETHICS follow. (The present "Principles" cover 48 sections and are spread over eight chapters.)

Preamble

These principles are intended to serve physicians, individually or collectively, as a guide to ethical conduct. They are not laws; rather they are standards by which a physician may determine the propriety of his own conduct. They are intended to aid physicians, in their relationships with patients, with colleagues, with members of allied professions and with the public, to maintain under God, as they have through the ages, the highest standards.

Section 1. The prime objective of the medical profession is to render service to humanity with full respect for both the dignity of man and the rights of patients. Physicians must merit the confidence of those entrusted to their care, rendering to each a full measure of service and devotion.

Section 2. Physicians should strive to improve medical knowledge and skill, and should make available the benefits of their professional attainments.

Section 3. A physician should not base his practice on an exclusive dogma or a sectarian system, nor should he associate voluntarily with those who indulge in such practices.

Section 4. The medical profession must be safeguarded against members deficient in moral character and professional competence. Physicians should observe all laws, uphold the dignity and honor of the profession and accept its self-imposed disciplines. They should expose, without hesitation, illegal or unethical conduct of fellow members of the profession.

Section 5. Except in emergencies, a physician may choose whom he will serve. Having undertaken the care of a patient, the physician may not neglect him. Unless he has been discharged, he may discontinue his services only after having given adequate notice. He should not solicit patients.

Section 6. A physician should not dispose of his services under terms or conditions which will interfere with or impair the free and complete exercise of his independent medical judgment and skill or cause deterioration of the quality of medical care.

Section 7. In the practice of medicine a physician should limit the source of his professional income to medical services actually rendered by him to his patient.

Section 8. A physician should seek consultation in doubtful or difficult cases, upon request or when it appears that the quality of medical service may be enhanced thereby.

Section 9. Confidences entrusted to physicians or deficiencies observed in the disposition or character of patients, during the course of medical attendance, should not be revealed except as required by law or unless it becomes necessary in order to protect the health and welfare of the individual or the community.

Section 10. The responsibilities of the physician extend not only to the individual but also to society and demand his cooperation and participation in activities which have as their objective the improvement of the health and welfare of the individual and the community.

Thus "The Principles of Medical Ethics" of the American Medical Association, upon which rest the conduct of physicians throughout this country, and perhaps the world, could become "The Ten Commandments of Medicine."

If these new PRINCIPLES are adopted by the House of Delegates, their brevity makes it

possible that they and the age-old Oath of Hypocrates could hang on the wall of physicians' offices together. They would be a daily reminder of the ideology, the concept of ethics, the framework under which all physicians strive to better themselves and the honored profession which they have chosen.

Opening Session

At the Monday opening session Dr. Elmer Hess, outgoing A.M.A. President, warned that the medical profession must be prepared to face an all-out drive by some labor groups for national compulsory health insurance. Dr. Dwight H. Murray, then President-Elect, told the House that general practitioners and specialists must guard against "any cleavage within our profession," and he urged strength through unity.

Dr. Lowell T. Coggeshall, special assistant to Secretary Marion B. Folsom of the U. S. Department of Health, Education and Welfare, assured the House that the over-all medical objectives of HEW are in accord with those of the A.M.A. A memorial plaque honoring the late Dr. Carl M. Peterson, secretary for 17 years of the A.M.A. Council on Industrial Health, was presented by Dr. Ross McIntire on behalf of the President's Committee on Employment of the Physically Handicapped. The Illinois State Medical Society presented a check for $164,940 to the American Medical Education Foundation.

Inaugural Program

Dr. Murray, in his inaugural address at the Tuesday evening ceremony in the Chicago Civic Oprea House, declared that "what we need most in medicine today is to find some way of combining modern scientific methods with the personal, friendly touch of the old-time family doctor." The inaugural program, which included the Bluejacket Choir of the U. S. Naval Training Center at Great Lakes, Ill., was telecast over Station WBKB in Chicago.

Election of Officers

In addition to Dr. Allman, the new President-Elect, the following officers were elected:

Dr. F. S. Crockett of Lafayette, Ind., Vice President; Dr. George F. Lull of Chicago, Secretary; Dr. J. J. Moore of Chicago, Treasurer; Dr. E. Vincent Askey of Los Angeles, Speaker, and Dr. Louis Orr of Orlando, Fla., Vice Speaker.

Dr. Julian Price of Florence, S. C., was reelected to the Board of Trustees, and Dr. Hugh Hussey of Washington, D. C., was named to succeed Dr. Allman. Dr. Robertson Ward of San Francisco was elected to the Judicial Council to succeed Dr. Walter F. Donaldson.

Reelected to the Council on Medical Education and Hospitals were Dr. Guy A. Caldwell of New Orleans and Dr. John W. Cline of San Francisco. Dr. Walter E. Vest of Huntington, W. Va., was named to succeed Dr. Louis A. Buie on the Council on Constitution and Bylaws.

Elected to the Council on Medical Service were Dr. Carlton Wertz of Buffalo, N. Y., to succeed himself, and Dr. J. F. Burton of Oklahoma City to succeed the late Dr. A. C. Scott, Jr. of Texas. Named for the three new places created on the Council on Medical Service were Dr. Thomas Danaher of Torrington, Conn.; Dr. R. M. McKeown of Coos Bay, Ore., and Dr. Lafe Ludwig of Los Angeles.

Respectfully submitted, J. D. Hamer, M.D.
Delegate to the A.M.A.
Phoenix, Arizona, June 23, 1956

BOARD OF MEDICAL EXAMINERS STATE OF ARIZONA

The Board of Medical Examiners of the State of Arizona at a regular meeting held April 21, 1956 issued certificates to practice medicine and surgery in this State to the following doctors of medicine:

BENSON, VICTOR GORDON, 407 16th Street, Yuma, Arizona.

BLINDE, OSCAR JOHN, Craig Hill, Ellensburg, Washington.

BOYD, JAMES WALLACE, 38 Westwood, Adrian, Michigan.

BULLINGTON, Robert Heyburn, 550 West Thomas Road, Phoenix, Arizona.

CROUCH, WARNER LATTA, 117 East Coronada, Santa Fe, N. M.

FITTIPOLDI, JR., JOHN, 3834 North 32nd Place, Phoenix, Arizona.

GEEVER, ERNEST DAN, 539 S. Hassayampa Dr., Prescott, Arizona.

GRECO, ROBERT, Morgantown, West Virginia.

HANSON, KENT OSCAR, Maricopa County Hospital, Phoenix, Arizona.

HOLSEY, WILLIAM F., 118-27 179th St., St. Albans, N. Y.

LINSLEY, LANCELOT EDWARD, 2491 Bainbridge, Eureka, California.

MOORE, LOUIS STONE, St. Joseph's Hospital, Phoenix, Arizona.

MUNHALL, HERBERT NICHOLAS, V. A. Hospital, Phoenix, Arizona.

ROADS, JOHN HORACE, 807 San Miguel, Phoenix, Arizona.

RUPPENTHAL, ARMOND J., 945 N. 12th Street, Milwaukee, Wis.

SPAULDING, RAYMOND CHARLES, 2430 East Sixth Street, Tucson, Arizona.

SPEELMAN, MERRILL EUGENE, Willcox, Arizona.

TUCHLER, MAIER I., 3020 Clay Street, San Francisco, California.

VIGIL, FRANK JOE, 102 West Merrell Street, Phoenix, Arizona.

VIVIAN, JOHN MARSHALL, 550 West Thomas Road, Phoenix, Arizona.

WILSON, LELAND BLAINE, 2250 Fairfield, Eureka, California.

WILSON, RICHARD VICTOR, 1603 N. Tucson Boulevard, Tucson, Arizona.

Organization PAGE

CIVICS – Norman Ross, M.D.

A MEDICAL COLLEGE IN ARIZONA!

There are eighty-six prime medical centers in the United States. The State of Illinois has one of these – Chicago. The State of Arizona has two such areas – Phoenix and Tucson. Our authority, Distribution of Physicians by Medical Service Areas, Bulletin No. 94, American Medical Association, Chapter 2, Page 19.

Arizona physicians with a program – Arizona educators with college curriculum adjustment – our State with organization – can have two medical schools in this day of desperate need of physicians. Let's be conservative and allow five years for the development of the first two years of this – **A Medical College in Arizona.**

In Maricopa County in 1945, the Maricopa County Medical Society Charities staffed Memorial Hospital and proved the local physicians' willingness and academic accomplishments by obtaining American Medical Association accrediting for advanced medical training in that institution. This was accomplished in one year.

Maricopa County physicians in 1951-1952 obtained American Medical Association accrediting at Maricopa County General Hospital for intern training and residency in general practice, and for two years of specialty training in surgery from "the College." Our large general local private hospital staffs are accredited.

Identical medical activities have resulted in accrediting for the Tucson hospital staffs. All of this is the result of organized groups within the framework of the Arizona State Medical Society and the local county medical societies.

In each of Arizona's populous areas, prime medical centers, there are well staffed activities that organized medicine, nationally, has recognized.

Leaders of industry and government – the group that organized and developed industry – the group that coordinated industry to government – are now decentralizing industry. They are, with the approaching accomplishment of their program, turning to a review of the nation's physician shortage. We suggest you read: "Every Other Bed" by Mike Gorman.

Medico-social activity of organized medicine in Washington and of the physician locally is reviewed in this book. Medicine's tradition, its schools, its behavior, its attitude toward a broad social program is presented and measured in **terms of public need – of national welfare and security.** Here is a call to appreciate values, to recognize adequate medical care, not only as a local, but a state and national problem.

The fact that medical schools are understaffed, that state institutions and programs **are begging for physicians,** in the face of over-supply and a huge reservoir of prepared, dedicated premedic students – who are waiting for admission to medical school – with many who may never hope to gain acceptance, is considered proof of the inadequacy of the present medical education concept and program.

Present means of selection and education in our medical educational programs under the guise of "the need to produce superior doctors," the finest local doctors, presents the prospect of continued scarcity – of few doctors – and for many Americans, but **not for want of money,** little or no medical care.

Here is a demand for medical educational definition and program, for speeding up of medical training, for new colleges in areas of plenty of qualified physicians. These men who have been faced with the problem of organizing and dispersing industry will see a need for the organizing and dispersing of educational facilities in medicine and in allied fields of health.

Arizona physicians can look to coming out of their respective private shells. They can take a just pride in their academic preparation and their social potential, in the quality of their colleagues.

Our state representatives and each of the 48 governors will welcome medical leadership. No political figure – Democrat or Republican – could object to the initiation of medical educational programs or facilities by men of medicine.

Arizona physicians will look to the need for medical care. We will act in the public interest. Arizona will accomplish two years of one medical college by 1961.

Woman's AUXILIARY

WOMEN'S AUXILIARY TO THE ARIZONA STATE MEDICAL ASSOCIATION

I. As President of the Women's Auxiliary to The Arizona State Medical Association, I wish to report the following activities for the year 1955-56.

1. American Medical Education Foundation.

All county auxiliaries have been made aware of the importance of the American Medical Education Foundation. Each county has worked hard to increase their contributions to this fund, within their own groups. The sum of $375.00 was raised throughout the state. All auxiliary members have been informed and urged to work on the special project of the Women's Division of the National Fund for Medical Education during Medical Education Week, April 22nd - 28th.

Subscriptions to the "80 Dimes Campaign" are to be solicited from lay persons and each member has been asked to seek at least one subscription. All auxiliaries have been asked to assist their county societies in handling publicity during Medical Education Week.

2. To-Day's Health

To date 599 subscriptions of To-Day's Health have been sold.

3. Bulletin

Each county and state officer has been urged to subscribe to "The Bulletin." Forty-four subscriptions have been bought in the state.

II. State Auxiliary Activities.

1. Program

Interesting and educational programs were planned around the theme "Active Leadership in Community Health." Program chairmen were urged to give a place to each committee chairmen on the program some time during the year. Civil Defense, Nurse and Allied Medical Services Recruitment, Mental Health and Legislation, were some of the most popular programs.

The report of the Conference of Presidents and President-Elects was given to all organized counties, and one unorganized county, during the year. The president and president-elect felt that giving this personally was immensely important in bringing out better relations between the state and county organizations. A school of instruction for all incoming officers and committee chairmen was held during the state convention.

2. Public Relations

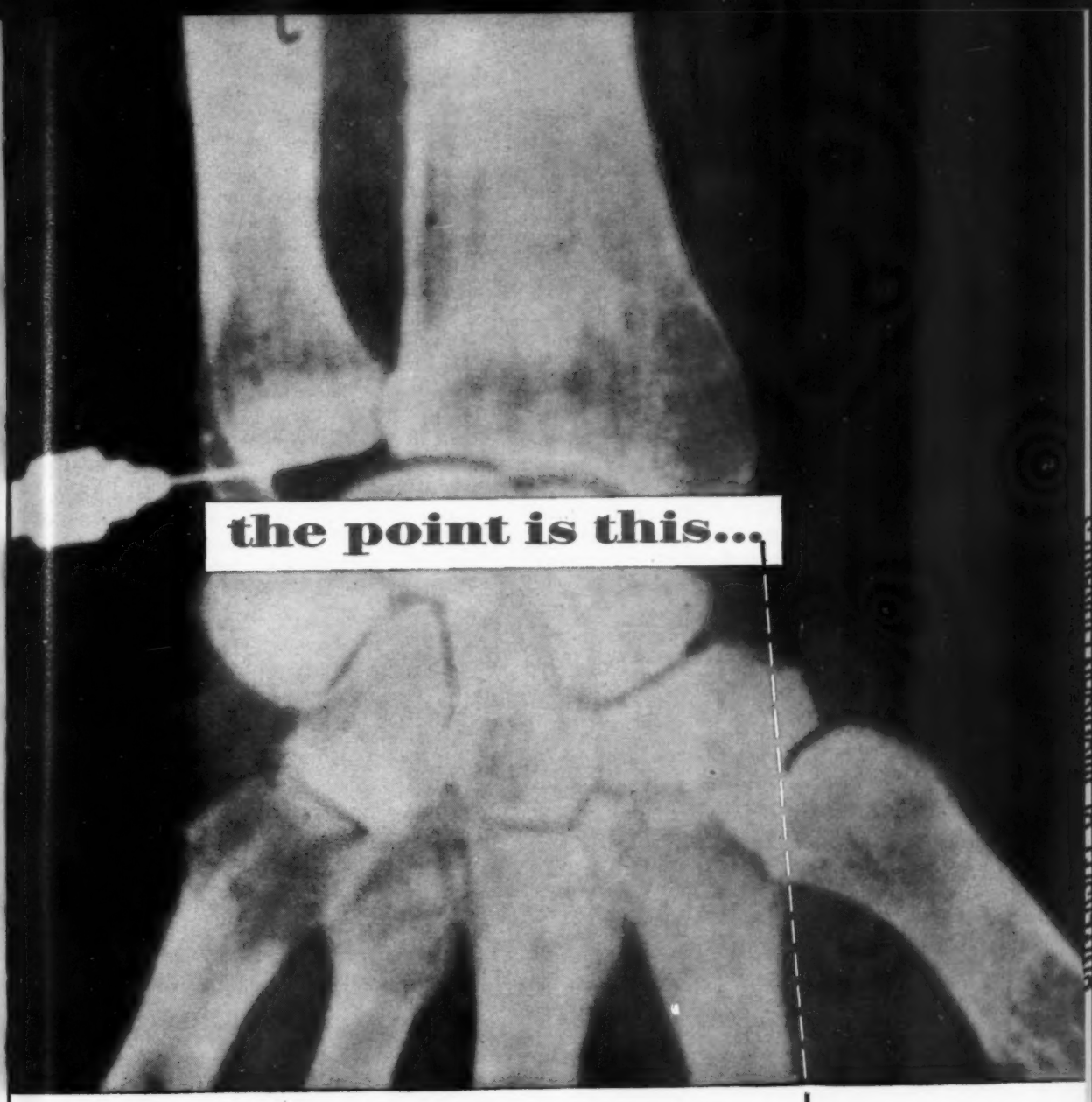
In public relations this year we were concerned with services to the communities in health. All counties participated in some form of health service. A radio health program was sponsored by one auxiliary, and two auxiliaries conducted audiometer tests, in public, county, and parochial schools. Two auxiliaries sponsored nurse recruitment, cancer, polio and To-Day's Health booths at their respective county fairs.

3. Community Service and Philanthropic Work

Each county participated in local solocitation of funds for service organizations such as Red Cross, March of Dimes, Cerebal Palsey. The Community Hospital in Prescott received over $4,000 from a charity ball sponsored by the Yavapai County Auxiliary. Maricopa raised over $1600.00 from a rummage sale. Of this sum, $1200.00 was given to the Child Guidance Clinic, and $175.00 to the Visiting Nurse Service. Direct donations of cash, food and gifts were made to individuals or organizations at Christmastime. Pima County secured a TV set for the County Hospital, and renewed subscriptions to magazines and newspapers. Six one-year subscriptions of To-Day's Health were given by one county to mothers with babies born on or near holidays. Many doctor's wives all over the state are members of hospital auxiliaries. This active participation in civic affairs is not only our obligation as doctor's wives, but as responsible citizens and members of our community.

4. Legislation.

For two years the Pima County Medical



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References: 1. Hollander, J. L., *Ann. New York Acad. Sc.* 61:511, May 27, 1955.
2. Hollander, J. L., *et al. J.A.M.A.* 158:476, June 11, 1955.

Auxiliary has been successfully laying the ground work for intelligent and effective participation in legislative efforts on a national scale. The Pima County Auxiliary assisted the Pima County Medical Society in securing a legislative expert and consultant, Dr. Marjorie Shearon. A schedule was planned for a whole week of speaking engagements for her in November. One talk was open to the public on the University Campus. Through the week, Dr. Shearon spoke before groups sponsored by dentists, pharmacists, and two Service Clubs. In Phoenix she spoke to a section of lay people, doctors, and auxiliary members. Every organized society had equal opportunity to engage the speaker. In January legislative letters were composed and sent from the Society's office, informing the membership about H.R. 7225 and its medical implications. This letter urged all doctors to write their Senators. The response to the request for letters was extremely good. We feel that this project accomplished what the Women's Auxiliary to the A.M.A. had earlier expressed the hope it would, namely;

a. An informed membership

b. Intelligent cooperation and consultation with our Medical Societies, and

c. An enlightened public.

As further evidence that much interest was aroused on the subject of Social Security, one of the local newspapers has carried a question and answer on Social Security, running two columns side by side, giving answers by a Mr. David of the Dept. of Health, Education and Welfare, and Dr. Marjorie Shearon. The questions asked were formulated by a citizen's group which in turn presented them to the editor of the local newspaper who sent them to the Dept. of Health Education and Welfare and Dr. Shearon for replies. These questions and answers, plus the letters to the editors certainly helped to inform the public.

5. Funds

Eight girls received loans from the Nurses Loan Fund this year. Two girls took partial loans of $200 00 each, while six had full loans of $400 00 each. Six girls enrolled at Good Samaritan School of Nursing, one in St. Joseph's and one in St. Mary's School. This year the Arizona Medical Association granted the Auxiliary an additional $800.00 in order to finance the large number of qualifying applicants for our Loan Fund. An anonymous gift of $400.00 was also received. To date, thirty girls have taken advantage of our Loan Fund and have used funds amounting to $8,300.00.

6. Publications

Each month articles have appeared in "Arizona Medicine." These articles written by state chairmen of the auxiliary, gave information about different phases of our program. Reports of The National Convention and Presidents and President-Elects Conference were also published. Two issues of "The Mail Box" were sent to every doctor's wife in the state, and to the editor of each state auxiliary. Over 800 copies per issue were distributed this year.

7. Organization.

Arizona has 5 organized counties with a total membership of 531, out of a possible 909 members. Members-at-large showed a 100% gain in membership. Fifty-one paid dues, this year. At convention this group was given recognition for the work they did in their own communities. One member, as chairman, gave their report.

8. Auxiliary Records

All reports of state officers, state committee chairmen, and county presidents are filed annually at the State Medical Association Headquarters.

III. ACTIVITIES JOINTLY WITH OTHER ORGANIZATIONS.

A. Nurse and Other Medical Services Recruit ment

Working closely with the Joint Committee on Careers in Nursing, which is made up of three members of the Medical Auxiliary, the Arizona Nurses Association, Association of Student Nurses, and the Nursing League, plans were laid for an over-all nurse recruitment program in the state. The District Presidents and Nurses worked with auxiliary members in each county, and the Careers Committee served in an advisory capacity and as distribution center for films and materials.

In November this committee maintained a Recruitment Booth at the State Fair; Maricopa Auxiliary helped staff the registration booth for the Mobile Chest X-Ray Unit, and also assisted in the transportation for the students who worked in the booth on Nursing Careers. Cost of this booth was shared by the Careers in Nursing Committee. Cost to the auxiliary was

$103.00. To-Day's Health was also given to interested spectators. Governor Ernest W. McFarland proclaimed the week of February 5th - 11th as "Nurse Recruitment Week." All training schools in Arizona held open house to girls interested in nursing. Many auxiliary members drove girls from outlying districts to the nursing schools. Over 106 girls attended open house at St. Mary's in Tucson.

The Maricopa County Recruitment Committee has carried out a very extensive program of recruitment for Allied Medical Services, including nursing, professional and practical, medical technology, X-Ray technique, dietitics, physical therapy, occupational therapy, medical records, and medical social work. The program was organized into a panel of speakers, one for each profession, who would speak ten to fifteen minutes. Each school in the area was notified and a convenient time arranged for the panel to appear. At least thirteen schools will have been visited by the end of the year.

A film of these allied medical services was made by the Maricopa Auxiliary to be used in some of the schools. This film was so outstanding that the National Association of Social Workers had a copy of the film made for their use. This committee also compiled a pamphlet including information regarding each career. It included prerequisites, training program, salary range, and places in the Southwest where training can be obtained. This pamphlet will be published by the National Association of Social Workers and distributed to all medical auxiliaries in the country as a guide.

One of the big projects of the Pima County Committee was to hold a fashion show of student nurse uniforms past and present, showing uniforms and giving information on these schools and about nurses training in general.

Mrs. Pearl Coulter, from the University of Colorado School of Nursing, was honored at a tea during the State League of Nursing Convention. Students, parents and councilors attended to gain information about collegiate schools for nurses.

In Arizona 11 Future Nurses Clubs are established with 2 more to be organized when the 2 new high schools are opened.

B. Mental Health

Considerable activity has developed this year in Arizona, both financially and educationally. One auxiliary gave $1200.00 for the benefit of The Child Guidance Clinic, and another auxiliary purchased 1,000 booklets for lay distribution during Mental Health Week. Films, "Search for Sanity" and "We The Mentally Ill" were shown during Mental Health Week. Another auxiliary sponsored a panel discussion by Junior High Students concerning group and personal problems pertinent to their age group.

Individual auxiliary members are represented on the Boards of:

1. Arizona Association for Mental Health
2. Maricopa and Pima County Associations for Mental Health.
3. Phoenix and Tucson Child Giudance Clinics.

C. Civil Defense

The object in Civil Defense has been a survey of the protected homes of auxiliary members in each county. Four organized counties reported a grand total of 106% participation, making the state percentage 40% (combining homes prepared and membership enlisted in Civil Defense activities). Six members in one auxiliary are active in the Ground Observer Corps.

D. Home Safety and Accident Prevention

One County assisted the State Safety Council on S-D Day.

It has been an honor and pleasure to serve as president of The Women's Auxiliary to the Arizona State Medical Association. I wish to thank all my officers and committee chairmen for their splendid work and loyal support; the national officers for their guidance; and the central office for its assistance.

MRS. ROY HEWITT